American Journal of Science and Learning for Development

ISSN 2835-2157
Volume 2 | No 5 | May -2023

Dyscyclatory Encephalopathy – Issues of Pathogenesis, Diagnosis, Differential Diagnosis at the Present Stage

M. B. Urinov [1], Allaev N. N. [2]

[1, 2] Bukhara State Medical Institute Department of Neurology

Abstract: *Timely full-fledged diagnosis of DE allows ensuring the choice of adequate tactics for the management of the patient, the selection of complex therapy covering the entire spectrum of pathogenetic and clinical variants of this disease and providing prevention of complications.*

Keywords: *discirculatory encepalopathy, brain circulation disorders.*

Chronic disorders of cerebral circulation are one of the most frequent pathologies. These include the initial manifestations of insufficient blood supply to the brain and dyscirculatory encephalopathy. DE is a chronic progressive insufficiency of blood supply to the brain, accompanied by diffuse and small-focal changes that occur due to various circulation disorders. At the same time, on the one hand, chronic cerebrovascular insufficiency is a risk factor for the development of ONMC, and on the other — the cause of a gradual increase in various neurological and mental disorders. The social significance of this pathology, because neurological and mental disorders in DE can cause a serious decrease in the quality of life and severe disability of patients. The main pathogenetic factors leading to the development of DE are arterial hypertension, stenosing lesion of the main arteries of the head, disorders of the hemostasis system, however, in the overwhelming number of patients, the pathogenesis of DE is multifactorial. Persistent increase in blood pressure is accompanied by damage to small cerebral arteries – microangiopathy, morphologically manifested by arteriosclerosis or lipogyalinosis. Changes in the arteries of the brain are detected in the form of foci of plasma, hemorrhages, zones and necrosis of the vessel wall with its thinning, elements of reparative and adaptive processes are simultaneously recorded in the form of hypertrophy of the muscular membrane with recalibration of the arteries, the formation of "hypertensive stenoses", up to complete obliteration of the vascular lumen [1]. Since arterial hypertension (AH) is the leading risk factor for progredient forms of cerebrovascular pathology (including DE), one of the most important issues is to determine the presence and nature of hypertension in a patient, and the most important parameters are such as the level of blood pressure, the degree of damage to target organs (heart, kidneys, brain) and etiology. Table 1 shows the classification of hypertension by blood pressure level in accordance with the recommendations of the Russian Society for Arterial Hypertension and the All-Russian Scientific Society of Cardiologists (3rd revision, 2008) [7].

Table 1. Classification of hypertension by blood pressure level (mmHg)

AD categories	SAD		DAD
Optimal	< 120	and	< 80
Normal	120 - 129	and/or	80 - 84
High Normal	130 - 139	and/or	85 - 89
• AG of the 1st degree	140 - 159	and/or	90 - 99
• AG 2 degrees	160 - 179	and/or	100 - 109

• AG 3 degrees	≥ 180	and/or	≥ 110
Isolated systolic hypertension *	≥ 140	and	< 90

Note: * ISAG should be classified into 1, 2, 3 degrees according to the SAD level.

This classification is of great importance, because it allows you to visually assess the severity of hypertension and, as a consequence, the risk of developing ONMC or progressive deterioration of cerebral hemodynamics. It is also extremely important to take into account the stage of hypertension and the presence of risk factors. At stage I of hypertension, there are no objective manifestations of target organ damage. In stage II, there is at least one of the instrumentally or laboratory detectable signs of target organ damage, such as left ventricular hypertrophy, retinal vasoconstriction, microalbuminuria, atherosclerotic lesion of the carotid, iliac and femoral arteries or aorta. In stage III, in addition to the listed signs, there are associated clinical conditions (ACS), which, according to the mentioned Recommendations, include ischemic and hemorrhagic cerebral stroke, as well as TIA. From a clinical point of view, it is necessary to take into account such conditions as cerebral hypertensive crises, hypertensive discirculatory encephalopathy, vascular dementia. Unfortunately, there is a combination of DE, TIA or hypertensive crises and stage I–II hypertension in the diagnosis of one patient, which indicates an underestimation of the patient's condition and the degree of risk of complications.

When assessing cardiovascular risk, the presence of another pathology also plays an important role. In particular, risk factors include high pulse blood pressure (in the elderly), age (men > 55 years; women > 65 years), smoking, dyslipidemia, diabetes mellitus and impaired glucose tolerance, family history of early CVD, abdominal obesity and a number of other conditions (see Recommendations). Risk stratification is carried out according to the parameters presented in Table 2.

Table 2. Risk stratification in patients with hypertension *

FR, POM and NW	**Blood pressure (mmHg)**			
	High normal 130-139/85-89	AG 1 degree 140-159/90-99	AG 2 degrees 160-179/100-109	AG 3 degrees > 180/110
No FR	Insignificant	Low additional risk	Average additional risk	High additional risk
1-2 FR	Low additional.** risk	Average additional risk	Average additional risk	Very high additional risk
≥ 3 FR, POM, MSiliSD	High additional risk	High additional risk	High additional risk	Very high additional risk
AKS	Very high additional risk	Very high additional risk	Very high additional risk	Very high additional risk

The clinical manifestations and course of encephalopathy depend on a number of factors, in particular on the individual characteristics of pathogenesis. In cases where the leading component of the pathogenesis of DE is hypertension, the predominance of step-like development of symptoms against the background of periodically developing hypertensive cerebral crises is the most characteristic. At the same time, intracerebral arteries with a diameter of 70-500 microns and the microcirculatory bed of the brain are mainly affected, the segmental nature of vascular lesions is typical. Developing vascular lesions are divided into primary – acute, repeated destructive changes caused by vascular crises (plasmorrhagia, fibrinoid necrosis with swelling of the wall and the development of acute hypertensive stenosis, isolated necrosis of the myocytes of the middle lining of the arteries, miliary aneurysms, wall rupture, thrombosis) and secondary – chronic reparative processes (arteriosclerosis, hyalinosis with thickening of the walls and narrowing of the lumen of the arteries up to obliteration), compensatory and adaptive changes (myoelastofibrosis, hyperelastosis, muscle-elastic "pillows" in the places of branch divergence, hypertrophy of the middle shell, proliferation of vessels of the microcirculatory bed). Thus, multiple diffuse and small focal changes in brain tissue, having different pathogenesis, localization, nature and prevalence, lead to the

formation of hypertensive angioencephalopathy. Patients with severe hypertension and repeated vascular crises are characterized by small deep (lacunar) cerebral infarcts, which are considered as a special form of ischemic brain damage. At the same time, lacunar syndromes such as "pure motor" stroke, "pure sensitive" stroke or "pure cerebellar" stroke usually develop first. With the repetition of such episodes, a lacunar state develops. This is a severe multi-focal pathology of the brain of an ischemic nature, formed with the development of multiple hypertensive small deep (lacunar) infarcts (for example, in patients with severe hypertension, multiple TIA), leading to a decrease in the mass and volume of the brain, expansion of the volume of the ventricles and subarachnoid spaces. Such patients are usually characterized by cognitive decline. When hypertension is combined with echeloned atherosclerosis, the formation of atherosclerotic small deep (lacunar) infarcts is characteristic, "phenotypically" similar to hypertensive ones, but having a different pathogenesis. In this case, it is not the perforating arteries themselves that are affected (as in hypertension), but the anterior, middle and posterior cerebral, as well as the extracranial sections of the carotid and vertebral arteries in various combinations. A decrease in blood flow in the perforating arteries is secondary.

In accordance with the peculiarities of pathogenesis and clinical course , DE is divided into the following forms:

I – Subcortical Atherosclerotic encephalopathy (SAE);

II – Multiinfarction condition (MIS);

III – Atherosclerotic discirculatory encephalopathy (ADE);

IV – Mixed forms: the most typical are SAE+ MIS, ADE+MIS;

V – Venous encephalopathy (VE).

Subcortical atherosclerotic encephalopathy (Binswanger's disease)

The morphological substrate is the lesion of small perforating arteries of the white matter of the brain with a diameter of less than 150 microns in the form of vascular wall hypertrophy, hyalinosis, sclerosis, accompanied by narrowing or complete closure of the vessel lumen, leading to diffuse damage to the brain substance (spongiosis, foci of incomplete necrosis, myelin decay, foci of encephalolysis).

Characteristic clinical manifestations are:

1. Progressive increase in cognitive impairment (decreased memory, attention, intelligence), reaching the degree of dementia in the last stages.
2. Gradual increase of emotional impoverishment, loss of interest in life, narrowing of the circle of interests.
3. Gradual increase of postural and locomotor disorders (primarily walking).
4. Pseudobulbar syndrome of varying severity.
5. The development of subcortical parkinsonlike syndrome is possible.
6. Mild and moderate paresis of the extremities is possible.
7. Violations of pelvic organ function control are gradually increasing

On CT, MRI, there is a decrease in the density of white matter, especially pronounced around the anterior horns of the lateral ventricles; the phenomenon of "leukoareosis" and multiple postinfarction cysts (after lacunar infarcts, sometimes clinically "mute") in the white matter and subcortical nodes.

Multiinfarction condition

The morphological substrate is a lesion of medium and large perforating arteries of the white matter of the brain with a diameter of 200-500 microns in the form of recalibration, hypertrophy of the vascular wall, hyalinosis, sclerosis, accompanied by narrowing or complete closure of the vessel

lumen, leading to diffuse and small-focal lesions of the brain substance. A combination of multiple infarcts of various genesis is characteristic. It develops mainly in patients with arterial hypertension, cardioembolism (with atrial fibrillation), narrowing of the lumen of intracerebral vessels as a result of their recalibration, as well as with various angiopathies and coagulopathies.

Characteristic clinical manifestations are:

1. Cognitive impairment, rarely reaching the degree of dementia;
2. Pseudobulbar syndrome;
3. Subcortical syndrome;
4. Cerebellar syndrome.
5. A step-like development of symptoms is characteristic

Signs	**Scores**
• Sudden development of dementia	2
• Step-like development	1
• Fluctuating development	2
• Disorientation at night	1
• Relative safety of personality	1
• Depression	1
• Emotional instability	1
• History of arterial hypertension	1
• A history of stroke	2
• Presence of atherosclerosis (ECG + UZDG)	1
• Focal neurological symptoms	2
• Pathological signs	2
With a total of 4 points or less – BA, 7 or more – DE	

In the management of patients with DE, the main directions are the prevention of disease progression and the treatment of major syndromes. The possibilities of pharmacological therapy of DE are extensive (Table 4), but for the selection of adequate therapy, it is necessary not only to ensure its pathogenetic validity, but also to select drugs that combine sufficiently high efficacy and good tolerability with long-term or course use.

Thus, timely full-fledged diagnosis of DE allows to ensure the choice of adequate tactics for the management of the patient, the selection of complex therapy covering the entire spectrum of pathogenetic and clinical variants of this disease and providing prevention of complications. Timely initiation of treatment allows not only improving the quality of life of the patient, but also significantly extending the period of his social activity.

Literature

1. Vereshchagin N.V., Morgunov V.A., Gulevskaya T.S. Pathology of the brain in atherosclerosis and arterial hypertension. M., Medicine, 1997, p. 288
2. Gudkova V.V., Stakhovskaya L.V., Kirilchenko T.D., etc. The use of Actovegin at different stages of treatment of patients with ischemic stroke. J. CONSILIUM MEDICUM/ vol. 9 / No. 8 from 22-25.
3. Gusev E.I., Belousov Yu.B., Boyko AN. and others . General principles of pharmacoeconomical research in neurology: Method. recommendations. M.; Publisher E. Razumova, 2003.-56 p.
4. Kamchatnov AVE., Chugunov A.V., Volovets S.A., Umarova H.Ya. Combined therapy of dis circulatory encephalopathy. Consilium medicum. 2005; 7 (8): 686-92
5. Kamchatnov P.R. Chronic disorders of cerebral circulation possibilities of metabolic therapy. - Breast cancer, volume 16, No. 5, 2008 - pp. 274-276.

6. Piradov M.A. Antiplatelet therapy in the prevention of recurrent ischemic strokes. - RMJ, 2003, volume 11, No. 12, pp. 696-698
7. Recommendations of the Russian Medical Society for Arterial Hypertension and the All-Russian Scientific Society of Cardiologists. - Appendix 2 to the journal "Cardiovascular therapy and Prevention" 2008; 7(6).
8. Jansen V., Bruckner G.V. Treatment of chronic cerebrovascular insufficiency using Actovegin forte pills. Russian Medical Journal. 2002; 10 (12): 1-4.
9. Collaborative meta-analysis of randomized trials of anti-platelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. BMJ 2002 Jan 19;324(7330):141.
10. Law M.R., Wald N.J., Rudnicka A.R. Quantifying effect of statins on low density lipoprotein cholesterol, ischaemic heart dis- ease, and stroke: systematic review and meta-analysis. BMJ. 2003 Jun 28;326(7404):1423.
11. Salomova N.K. Features of the course and clinical and pathogenetic characteristics of primary and repeated strokes //Central Asian Journal of Medical and Natural Science. 2021.-C. 249-253. (14.00.00.№).
12. Salomova N. K., Rakhmatova C.N. Kaita takrorlanuvchi ischemic va hemorrhagic stroke bemorlarni erta rehabilitation of kilishni optimallashtirish // Journal of Neurology and Neurosurgical Research. 2021y. 71-76 bet,(14.00.13. №14.).
13. Salomova N.K // Risk factors for recurrent stroke// Polish journal of science N52(2022). 33-35.
14. Salomova N.Q //The practical significance of speech and thinking in repeated stroke// ScienceAsia 48 (2022): 945-949.