PREVENTION AND TREATMENT OF COGNITIVE DISORDERS IN PATIENTS WITH ARTERIAL HYPERTENSION

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Abstract

The article contains systematized literature data concerning prophylactic and rational approaches of antihypertensive treatment of cognitive disturbances in patients with arterial hypertension (AN). These approaches are based on the results of multi-center, randomized studies in this patient group. It was shown that applying in time antihypertensive drugs having certain cerebroprotector abilities and correction emotional and metabolic disorders make positive influence on clinics and prognosis of AN, prevent the progressing of cognitive disturbances. It was also shown that in patients with AH associated with cognitive disturbances long-term therapy with angiotensin II antagonists, angiotensin-converting enzymes inhibitors and dihydropyridine calcium blockers make positive influence on clinics and prognosis of AN and could in some cases predict the progress of cerebrovascular disturbances.

Key words: arterial hypertension, cognitive disorders, antihypertensive therapy.

It is known that high blood pressure (hypertension) is an important risk factor for development of stroke and chronic brain circulatory disorders, which present with cognitive disorders in a lot of cases. Outcomes of cognitive disorders include declined quality of life and development of various forms of vascular dementia. As a rule, development of dementia results in permanent disability, followed by complete loss of competence, which is characterized with significant socio-economic damage [1]. Majority of dementia cases are associated with two different pathologies: Alzheimer's disease and the so-called vascular dementia. Less frequent causes include endocrine system diseases, metabolic disorders, and some infections [2].

From the standpoint of cardiovascular diseases, vascular dementia is more important. Dementia of vascular genesis implies declined cognitive functions as a result of ischaemic or hemorrhagic brain damage caused by a primary cerebrovascular or cardiovascular disease [3]. Such type of dementia may be caused by both acute or chronic brain circulatory damage. Cognitive disorders are early signs of chronic vascular brain damage [4]. As evidenced by recent research, hypertension is an independent risk factor for development of cognitive dysfunction

in all age groups; high blood pressure in adolescents may be accompanied by a number of cognitive function disorders, specifically mathematic ability and creativeness [5]. Long-term observation of middle-aged individuals suffering from hypertension demonstrates that they are more prone to develop cognitive disorders and dementia at elderly age, compared to individuals with normal blood pressure. Level of blood pressure at age 50 correlates with thinking ability at age 70, as lower BP is associated with less deteriorated cognitive functions. Here, hypertension acts as a catalyst for development of stroke and other changes in brain vessels and decreases the age at which dementia threshold is reached [6].

Association between hypertension and risk of dementia is obvious from the standpoint. Probability of dementia within a year after stroke in hypertension patient is 20-30%. On average, stroke in hypertension patients increase risk of dementia by two-fold as a minimum [7].

Possible mechanisms of development of cognitive disorders in hypertension include leuko-areosis and asymptomatic lacunar infarcts, which lead to development of the syndrome of disconnection between cortical brain and subcortical ganglions and subcortical lobar dysfunction [3]. Leukoareosis presents with bilateral focal and

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diffuse sites of decreased density in white matter, determined by computer tomography (CT) and T1-weighted magnetic-resonance imaging (MRI). Clinically, leukoareosis is often of asymptomatic nature, however, in clinically ill patients it may cause deterioration of cognitive functions, gradually progressing into dementia [6]. Majority of infarcts in hypertension patients localize in functionally "silent" parts of brain (sites of white matter of hemispheres), which explains asymptomatic nature of the process and which are accidentally found in MRI. In patients with multiple "silent infarcts", decreased cognitive functions are observed and they are at higher risk of developing dementia within next several years [7].

Other possible pathway of formation of cognitive disorders in hypertension is shortening of time interval until clinical neurodegenerative disease and progression of this process. In hypertension patients, brain damage is often combined with Alzheimer's disease. Prevalence of "mixed" (vascular and degenerative) dementia is not less than 20%, which is significantly higher than what would be expected in random combination of these two disorders. Until 1996, it was thought that hypotension is a predisposing factor for Alzheimer's disease. However, later it was demonstrated that Alzheimer's disease is mainly observed in patients who had hypertension during 9-15 years prior to the onset of Alzheimer's disease [8]. Currently, hypertension is considered as a factor accelerating realization of genetic predisposition towards degenerative process. Periodic or chronic cerebral ischaemia occurring in hypertension causes brain hypoxia and neurons of hippocampus in temporal lobe are most sensitive to oxygen insufficiency. In hypertension patients, who have early signs of Alzheimer's disease, development of cerebral infarct triggers further deterioration of cognitive functions and may lead to clinically evident dementia. Even "silent" cerebral infarcts may provoke degenerative changes in brain (senile patches, neurofibrillar glomerules), characteristic for Alzheimer's disease [8].

Thereby, development of dementia in hypertension may be a result of previous strokes,

including clinically "silent" infarcts, white matter damage (leukoareosis), as well as acceleration of degenerative processes in brain cortex. Currently, it is established that hypertension and atherosclerosis are not only risk factors for stroke and vascular dementia, but for Alzheimer's disease as well [9].

Finally, effectiveness of anti-hypertensive therapy in prevention of dementia and improvement of cognitive function serves as an indirect proof of association between hypertension and dementia. In elderly individuals with hypertension, cognitive disorders are identified more frequently when no adequate treatment is in place [10].

Prevention of dementia and cerebral protection in anti-hypertensive therapy. Large-scale studies conducted in last decade suggest that adequate anti-hypertensive therapy may serve as one of the promising prevention approaches for cognitive disorders [11,12,13]. Anti-hypertensive therapy for prevention of vascular brain disorders builds on main principles referenced by physicians: continuity, consistency, use of pharmaceutical drugs of modern classes. However, data collected in clinical trials allows identifying certain drugs with primary cerebral protection features, which serves as a basis for recommending these products as choice of treatment for patients who are at high risk of developing cognitive disorders. Effectiveness of anti-hypertensive therapy for prevention of dementia has been shown in a number of studies. First large study looking at this aspect of antihypertensive therapy was Syst-Eur study [10], where 2800 enrollees were randomized into two groups depending on administered therapy: calcium antagonist (CA) nitrendipine and placebo. One of the major findings of the study was decline in risk of dementia by 55% in therapy group (from 7.4 to 3.3 cases per 100 patients per year). It is important to note that such effect was similarly observed in AD and post-stroke dementia. PROGRESS study [11] was the next step in studying abovementioned effect. In this study, possible use of angiotensin converting enzyme (ACE) inhibitors and their combination with diuretics in prevention of recurrent stroke was demonstrated. In addition,

in active therapy group, decrease in total number of dementia cases by 12% was statistically insignificant, whereas statistically significant decline in dementia cases associated with stroke by 34% was noted.

Recent clinical trials studying prevention of dementia have targeted relatively novel classes of antihypertensive drugs, namely angiotensin converting enzyme II (ACE II) inhibitors. Significant improvement of cognitive functions was demonstrated upon treatment using losartan, compared to hydrochlorothiazide [14]. SCOPE [12] - (The Study on Cognition and Prognosis in the Elderly), the first large study specifically designed for evaluating cognitive functions was an international, multi-center placebo-controlled study of the effect of candesartan on main cardiovascular events and cognitive functions in elderly patients with mild and moderate hypertension. In SCOPE study, dementia occurred at a statistically significant rate in patients with low cognitive functions (24-28 points per Short Mental Status Evaluation Scale - SMSES) prior to therapy, compared to patients with high cognitive functions (29-30 points per SMSES). In the group of patients with low cognitive functions less significant decrease was noted upon therapy with candesartan, as compared to placebo, which demonstrates effectiveness of anti-hypertensive therapy using candesartan regarding inhibition of progression of cognitive disorders in elderly patients with hypertension. Relatively significant results were obtained in a recent MOSES [13] trial, studying cerebral protection in hypertensive patients. Patients who had stroke in their history were enrolled in this study. Therapy group was administered ACE II receptor antagonist eprosartan 600 mg/day. Unlike in PROGRESS study, here control group received ACE nitrendipin, instead of placebo, which has already proven to be effective in prevention of cerebral complications of hypertension. Major outcomes of the study included decrease of overall mortality by 20% among all cardiovascular and cerebrovascular disease cases; decrease of recurrent strokes and associated disorders (transient ischaemic attacks) by 25%; and decrease of primary cardio-

vascular complications by 30%, as well as comparable incidence of dementia in both groups.

No significant deterioration of cognitive functions was noted for the total duration of treatment both in eprosartan and nitrendipin groups, which may be considered a positive outcome, taking into account that patients became older by the end of the four years of observation period and some of them had stroke. Comparison of cognitive functions among two groups did not reveal any significant differences at the end of observation period. Considering the results of previous studies, which had demonstrated the effectiveness of eprosartan in prevention of cognitive disorders in hypertension patients, similar effectiveness may be expected from eprosartan, as well.

It is assumed that, anti-atherogenic effect of ACE inhibitors and ACE II receptor antagonists occur due to their interaction with rennin-angiotensin-aldosterone system (RAAS). RAAS not only plays an important role in pathogenesis of hypertension, but is also involved in genetic, humoral, and cellular pathways of atherosclerosis formation [15]. Activation of RAAS triggers oxidative stress and endothelial dysfunction, stimulates expression of low density lipoprotein receptors, increases release of anti-inflammatory cytokines and adhesive substances, enhances platelet aggregation, and is associated with proliferation of smooth muscle cells, increased level of calcium in these cells and increased production of collagen. In research studies, it was demonstrated that high level of angiotensin II in blood negatively impacts training of mice, as well as learning and memorizing new habits by them. Use of ACE II antagonists and ACE inhibitors may improve process of training in such cases, apparently due to inhibition of RAAS activity, engaged in regulation of behavioral reaction. Reports have been made that, unlike ACE inhibitors not penetrating brain tissues (enalapril and imidapril), those with such ability (captopril or perindopril) inhibit decline in cognitive functions in patients above 65 suffering from mild or moderate Alzheimer's disease [16].

However, whether or not priority should be given to certain anti-hypertensive drugs in pre-

vention of cognitive disorders in hypertensive patients requires further investigation.

Neurologic aspects of optimizing anti-hypertensive therapy. Neurologic complications of hypotensive therapy are primarily seen in elderly patients, especially upon presence of cerebrovascular disease and considerable stenosis of pre-cerebral (carotid and vertebral arteries) and cerebral arteries [17]. In individuals with normal range of blood pressure lower threshold of autoregulation of cerebral circulation is typically about 60 mmHg based on average hemodynamic arterial pressure. In patients with tedious hypertension lower limit of autoregulation of cerebral circulation is significantly higher and may reach 90-100 mmHg based on findings of average hemodynamic arterial pressure. The higher is typical blood pressure, the higher will the upper level of autoregulation of cerebral circulation be. Therefore, in patients with tedious and high arterial hypertension, hypo-perfusion of cerebral circulation may occur in relatively normal values of BP, especially, when efforts are made to quickly normalize BP [18]. Studies of brain circulation in patients with cerebrovascular disorders demonstrate that brain hypo-perfusion occurs upon lowering BP by 10-20% compared to "working BP" values [19]. This is associated with structural/functional changes in small brain arteries and arterioles, as a result of which compensatory dilation of small intracerebral vessels in response to decreased BP is not as high compared to normal BP, to support adequate cerebral circulation. Therefore, a conclusion may be drawn that no single level of BP may be targeted in patients who already have cerebral pathology. Presence of hemodynamically significant atherosclerotic occlusion of cerebral arteries is another important factor undoubtedly affecting selection of target level of BP. In addition, in these patients BP values correlate with risk of stroke and the least number of cerebrovascular events is registered at SBP of 140-150 mmHg. In cases of bilateral stenosis of carotid arteries > 70%, significant decrease of brain perfusion, as well as inverse correlation between level of SBP and risk of stroke are observed, where the least risk of stroke is registered at SBP values of 160-170

mmHg [20]. We should probably agree that in practice, using presented values of SBP as a treatment target for patients with cerebrovascular disorder is problematic, as there is no real-life data regarding status of cerebral perfusion and brain changes in the course of anti-hypertensive therapy. Markers of hyporeactivity of cerebral vessels, i.e., predictors of risk of cerebral hypoperfusion in cases of inadequate BP lowering, have been developed for optimization of approaches to treatment of hypertension in coinciding cerebrovascular disease: presence of clinical signs of extrapyramidal and/or pseudobulbar syndrome, left ventricular hypertrophy (LVH), and ischaemic heart disease (IHD). Special attention should be given to comparability of above-mentioned criteria, e.g., typical signs of pseudobulbar or extrapyramidal syndrome coincides with LVH or focal (or diffuse) changes in brain tissue. This allows using clinical symptoms as a guide in cases of non-feasibility of instrumental examination of patient [21].

Target level of BP in patients with cerebrovascular disorder in addition to hypertension should be selected considering compensatory capacity of cerebral hemodynamics. Above-listed markers of cerebrovascular hyporeactivity may serve as indicators of significant damage in compensatory ability. SBP and DBP may be lowered by 20% and 15%, respectively, upon retained adaptation reserve of cerebral circulation. SBP and DBP should not be lowered by more than 15% and 10%, respectively, compared to baseline level, if significant damage is observed in regulation of cerebral circulation, i.e., when above-listed markers are identified [22]. It should be noted that, such approach to lowering BP substantially differs from commonly-accepted treatment approach used in uncomplicated hypertension with the purpose of primary prevention of cerebrovascular disorders, where achieving target level of BP is a priority. Apparently, adequate relative reduction of BP is implied here, which corresponds to autoregulation capabilities of brain without further deterioration of cerebral perfusion [23]. It is important to note that, although small, however long-term (3-5 years) consistent lowering of BP in patients with cere-

brovascular disorders significantly improves long-term outcomes [24].

Self-treatment of hypertension often causes significant decrease of BP. Such may also occur upon treatment of increased blood pressure, where use of long-acting anti-hypertensive drugs does not provide immediate effect, but later significantly decrease blood pressure. In some cases, decreased BP may be observed upon intake of additional anti-hypertensive drugs without checking BP, when patient does not feel well, which is not always associated with high BP, but may be caused by low BP, as well [25].

In majority of cases hypo-perfusion caused by significant decrease of BP presents with dizziness and general weakness, which quickly resolves upon normalization of BP. Such conditions may occur asymptotically and be identified in ultrasonic dopplerography or magnetic-resonance spectroscopy in form of decreased cerebral circulation. Apparently, these hypo-perfusion cases may serve as one of the causes of "silent" strokes, which are frequently found in hypertensive patients in computer tomography of head [26]. Initial administration of smaller doses of hypotensive drugs, allowing only slightly decreasing BP is recommended for patients with cerebrovascular disorders. Relatively high values for BP, such as 140-160 mmHg for SBP and 80-90 mmHg for DBP may be targeted in presence of significant stenosis or occlusion of carotid and vertebral arteries, especially, when surgical intervention is not an option [27].

Finally, vast majority of patients with hypertension-related cerebrovascular disorder have signs of damage in another important target organ, being heart. Research studies conducted in last decade reveal close relationship between cardiac and brain pathologies, occurring as a result of various cardiovascular diseases. Thorough evaluation of patients suffering from cerebrovascular disorders allows revealing changes in heart in 75-80% of cases [28].

It has been established that, chronic heart disease (CHD) is identified in about 80% of hypertensive encephalopathy patients, where over half of the cases have an asymptomatic course and are identified in Doppler echocardiography only.

In addition, even initial stages of CHD (I-IIA) are characterized with dependence of circulation in vertebral arteries from contractive activity of heart [29]. This aspect should not be ignored upon selection of anti-hypertensive therapy.

Cardiac fibrillation extremely increases risk of stroke (about 12% per year). Latest recommendations include administration of indirect anticoagulants in all patients with atrial fibrillation, considering risk of recurrent thromboembolic complications [30]. In cases excluding use of anticoagulant therapy, drugs with platelet anti-aggregation activity may also be selected for secondary prevention of strokes of cardiac origin [31].

Taking into account that, cardiovascular remodeling developing in hypertension and thickening of arterial intima and media layers are also associated with dementia, optimization of heart activity becomes an important aspect, especially in treatment of CHD in hypertensive patients. However, primarily elderly age of patients suffering from CHD requires cautions in selection of treatment, especially β -adrenoreceptor blocking agents. SENIORS study [32] has clearly demonstrated effectiveness and safety of a β -adrenoreceptor blocking agent nebivolole in treatment of elderly patients suffering from CHD. Considering strong hypotensive characteristics of nebivolole, use of this drug in hypertension patients with CHD provides simultaneous solution for antihypertensive therapy, as well [33].

Neuropsychology studies among hypertension patients reveal that, consistent normalization of BP via anti-hypertensive therapy results in improvement of cognitive functions even during relatively short-term (3-6 years) treatment. Results of long-term observations (9-12 years) of elderly patients suffering from hypertension also show that decreasing systolic blood pressure may prevent cognitive disorders; in addition positive effect of normalization of BP is noted in group of patients who did not have stroke during the whole period of observation [34].

Treatment of headache, stress, and various psycho-emotional disorders in patients with hypertension is very important, where combina-

tion of anti-hypertensive agents with anti-depressants or anxiolytic drugs may be useful [35].

Correction of metabolic disorders, especially adequate hypolipidemic therapy also plays an important role both for primary and secondary prevention of stroke. All large studies conducted until today [36,37] have provided evidence regarding effectiveness of statins in decreasing ischaemic stroke-related mortality, as they provide systemic effect on various arteries, including carotid arteries.

Conclusion. The following conclusion may be withdrawn considering aforementioned: normalization of BP via use of anti-hypertensive therapy is one of the effective approaches in prevention of dementia and mild cognitive disorders in hypertension patients. From this standpoint, anti-hypertensive therapy in elderly patients who are at high risk of stroke and dementia in the upcoming years is especially important. Appearance of even slight cognitive disorders in hypertensive patients requires more strict control over BP, although rapid and significant lowering of BP should be avoided in such instances, due to possibility of declined cerebral circulation and deterioration of cognitive functions. Use of agents that support routine daily profile of BP

should be preferred, as they do not adversely affect metabolism and do not cause rapid decrease of BP. However, whether there is a necessity to achieve a single level of BP via anti-hypertensive therapy is unclear. Approach to level of decreasing BP for prevention of cerebrovascular disorders, where achieving a target level of BP is needed, significantly differs from techniques used for treatment of hypertension which co-incides with cerebrovascular disorder. In such cases, apparently, adequate relative reduction of BP should be targeted, which would correspond to auto-regulatory capacities of cerebral circulation and would not lead to further deterioration of cerebral perfusion. Reasonable combination of anti-hypertensive agents, with the absolute inclusion of those with proven efficacy in evidence-based medicine, is the most appropriate approach to prevention of cognitive disorders in hypertensive patients. Available data suggests that, calcium antagonists, angiotensin II receptor antagonists, and ACE inhibitors possess independent neuroprotective ability and may have certain advantages over other classes of anti-hypertensive agents in prevention of cognitive disorders in patients suffering from hypertension.

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XÜLASƏ

ARTERİAL HİPERTENZIYALI XƏSTƏLƏRDƏ KOQNİTİV POZUNTULARIN PROFİLAKTİKƏ VƏ MÜALİCƏSİ

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Təqdim olunan icmalda arterial hipertenziyalı (AH) xəstələrdə koqnitiv pozuntuların korreksiyasının profilaktikası və bu məsələyə rəşional yanaşmaları işıqlandıran ədəbiyyat göstəriciləri sistemləşdirilmişdir. Qeyd olunan yanaşmalar çoxmərkəzli, randomizəolunmuş tədqiqatların göstəricilərinə əsaslanmışdır. Göstərilmişdir ki, müəyyən serebral protektor xassələrə malik antihipertenziv preparatların vaxtında qəbul olunması və yanaşı gedən nevroloji, ürək-damar sistemi xəstəliklərinin və metabolik pozuntuların müalicə olunması AH olan xəstələrdə koqnitiv pozuntuların profilaktikasında müəyyən pozitiv rol oynayır. Həmçinin göstərilmişdir ki, AH olan xəstələrdə sinir- damar pozuntularının profilaktikasında II angiotenzin reseptorlar blokatorları, angiotenzinçevirici ferment inhibitorları və kalsium antoqonistləri başqa sinif antihipertenziv preparatlara nisbətən müəyyən üstünlüklərə malikdir.

Açar sözlər: arterial hipertenziya, koqnitiv pozuntular, antihipertenziv müalicə.

РЕЗЮМЕ

ПРОФИЛАКТИКА И ЛЕЧЕНИЕ КОГНИТИВНЫХ НАРУШЕНИЙ У БОЛЬНЫХ АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИЕЙ

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Известно, что артериальная гипертензия (АГ) является важнейшим фактором риска развития инсульта и возникновения хронических форм нарушений мозгового кровообращения, одним из самых значимых симптомов которого являются когнитивные расстройства. Последствием когнитивных нарушений можно считать снижение качества жизни пациента и формирование в последующем различных типов сосудистой деменции. Суммируя вышеизложенное, можно сделать следующие основные выводы по анализируемой проблеме: нормализация артериального давления (АД) в результате антигипертензивной терапии представляет одно из эффективных направлений предупреждения деменции и более легких когнитивных нарушений у пациентов с АГ. Особое значение в этом аспекте имеет антигипертензивная терапия у пожилых пациентов, имеющих высокий риск развития инсульта и деменции в ближайшие годы. Имеются основания предполагать, что антагонисты кальция, блокаторы рецепторов ангиотензина II и ингибиторы АПФ имеют самостоятельный нейропротективный эффект и могут иметь определенные преимущества перед другими классами антигипертензивных средств в профилактике когнитивных расстройств у пациентов с АГ.

Ключевые слова: артериальная гипертензия, когнитивные расстройства, антигипертензивная терапия.

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