IJIAET International Journal of Innovative Analyses and Emerging Technology

| eISSN: 27924025 | http://openaccessjournals.eu | Volume: 1 Issue: 4

Assessment of Pathogenetic Factors of Cerebrovascular Pathology in Type 2 Diabetes Mellitus

Davronova Khilola Zavkiddinovna

Department of Neurology, Bukhara State Medical Institute

ABSTRACT: Cerebrovascular diseases are one of the most pressing medical and socio-economic problems. In the structure of total mortality, cerebrovascular disease accounts for 21.4% of accidents and disability after stroke is 3.2 per 10,000 people, making it the leading cause of primary disability. Diabetes is one of the most important risk factors for stroke. Diabetes is now a global medical and social problem, and its growth rate has reached the level of a global epidemic. This article discusses the pathological progress that exists between these two diseases.

Keywords: Diabetes, cerebrovascular disease, microcirculation, dementia, neurodegenerative process.

Currently, there is no doubt about the role of type 2 diabetes mellitus (type 2 diabetes) in the occurrence of vascular pathology of the brain. Having type 2 diabetes increases the likelihood of developing a stroke by 2-6 times. In the MRFIT (The Multiple Risk Factor Intervention Trial), the risk of death from stroke among patients with diabetes was 2.8 times higher than in patients without diabetes, while the risk of death from ischemic stroke was 3.8 times higher. from subarachnoid hemorrhage - by 1.1 times and intracerebral hemorrhage - by 1.5 times [1]. The main mechanisms of the development of cerebral vascular pathology in type 2 diabetes are atherosclerotic lesions of large and small cerebral arteries, microcirculation disorders [2-4].

To systematize vascular diseases of the brain, it is now accepted to use the International Classification of Diseases and Problems Associated with Health, 10th revision (ICD-10), where cerebrovascular diseases are subdivided into the following main pathological conditions.

- Subarachnoid hemorrhage
- Intracerebral hemorrhage (hemorrhagic stroke)
- Brain infarction (ischemic stroke)
- Stroke, unspecified as hemorrhage or heart attack
- Other cerebrovascular diseases

progressive vascular leukoencephalopathy

hypertensive encephalopathy cerebral ischemia (chronic)

- Consequences of cerebrovascular diseases
- Transient transient cerebral ischemic attacks (attacks)
- Moderate cognitive impairment and vascular dementia.

In addition to cognitive disorders and vascular dementia, a separate class (class V) includes Alzheimer's disease, or Alzheimer's dementia, which develops as a neurodegenerative process. At the same time, a number of authors note that there is a direct link between Alzheimer's disease and vascular disorders, systemic atherosclerosis [5].

An important predictor of morbidity and mortality associated with acute cardiovascular diseases is the hemoglobin level. When 5102 patients with type 2 diabetes were observed in the UK for an average of 5 years (UKPDS study), it turned out that 674 patients developed myocardial infarction (351 with a fatal outcome), and 199 had cerebral stroke, and 35 had repeated (48 - fatal). The hemoglobin peak (HBA1c) level was closely correlated with the incidence of stroke, while an increase in the HBA1c concentration by 1% was accompanied by an increase in the risk of cerebral stroke by 17% [6].

It has been established that not only hyperglycemia is a risk factor for cardiovascular diseases, but also insulin resistance is closely associated with an increased risk of cerebral stroke [7, 8]. Violation of insulin sensitivity is more often observed in young patients with overweight; it is associated with lacunar cerebral infarction and stroke with persistent neurological deficit [9].

Insulin is directly involved in the implementation of a number of cognitive functions, and disorders of its metabolism are accompanied by the emergence of a number of syndromes of impaired neurological functions and cognitive disorders [10, 11]. Insulin is involved in the regulation of the production of protein - the precursor of amyloid and the

ISSN 27924025 (online), Published under Volume: 1 Issue: 4 in September2021

product of its metabolism - amyloid beta - the main component of amyloid deposits. Insulin regulates phosphorylation of tau protein, which is the basis of neurofibrillary formations in patients with cognitive impairments [12].

It is believed that insulin is involved in the exchange of beta-amyloid and reduces the rate of its degradation processes (inhibiting the corresponding enzymes), thereby contributing to the accumulation of abnormal proteins in the brain tissue and the progression of Alzheimer's disease. To a certain extent, this is confirmed by the high incidence of insulin resistance in patients with Alzheimer's disease [13].

Over the past two decades, the concept of risk factors for cerebrovascular complications has expanded significantly. These factors, along with traditional ones (arterial hypertension, dyslipidemia, smoking), began to include an increase in the thickness of the intima / media of the common carotid arteries, asymptomatic stenosis of the main arteries of the head, markers of inflammation, hyperfibrinogenemia and other disorders in the hemostatic system [4, 15].

Clinical features of cerebrovascular diseases in type 2 diabetes In addition to the fact that type 2 diabetes is a risk factor for cerebrovascular diseases, disorders of carbohydrate metabolism largely determine the clinical features and severity of neurological symptoms in patients with cerebrovascular accidents. It was found that a high level of glucose in the acute stage of the disease is significantly associated with a higher mortality rate and severe disability in patients with ischemic stroke, especially in the case of extensive infarction or total cerebral ischemia [16]. Fasting glucose levels exceeding 6.1 mmol / L and glycemic levels at any time during the day exceeding 7.2 mmol / L at the time of hospitalization are independent significant risk factors for death and insignificant recovery of neurological functions [17]. The likelihood of death due to stroke in patients with type 2 diabetes is as high as in patients with recurrent stroke, and the presence of concomitant cardiovascular diseases significantly increases the likelihood of death from stroke [8].

It is noteworthy that hyperglycemia in patients on admission to hospital for subarachnoid hemorrhage is also associated with higher mortality and less complete recovery of neurological functions [9].

It should be noted that the clinical features of ischemic stroke are also closely associated with the glucose level at the time of its development. There is evidence that the effectiveness of selective thrombolysis using tissue plasminogen activator (tPA) was lower in patients with initially higher blood glucose levels (more than 7.8 mmol / L) [10].

The presence of type 2 diabetes predisposes to the development of certain types of stroke [1]. Analysis of the results of observation of 4064 patients with stroke for an average of 5.3 years made it possible to establish that intracerebral hemorrhages were relatively less common in patients with type 2 diabetes compared with individuals with normal carbohydrate metabolism, and lacunar strokes caused by the pathology of small arteries were significantly more frequent. and atherothrombosis, while there were no significant differences in the incidence of cardioembolic stroke [9]. At the same time, in another series of observations, which included 937 patients with type 2 diabetes out of a total of 4537 patients with stroke from 7 European countries, an association was established between the presence of type 2 diabetes and a more pronounced neurological deficit by the end of the 3rd month of the disease [3]. In addition, these patients had a more severe disability. The authors noted the prevalence of lacunar infarctions in patients with disorders of carbohydrate metabolism, clinically manifested by paresis of the extremities and dysarthria.

At present, the problem of cerebral infarctions, not accompanied by a clear focal neurological deficit of transferred infarctions in the deep or periventricular parts of the white matter, caused by ischemia in the areas of blood supply of small perforating arteries, is of great interest. There is evidence that cerebral infarctions, despite the absence of clear clinical manifestations, are closely associated with the unfavorable course of various forms of cerebral vascular pathology, in particular, with repeated strokes and progressive vascular dementia [4]. The risk of developing cerebral infarctions increases sharply with a combination of type 2 diabetes and arterial hypertension, and the presence of multiple postischemic foci is observed in such patients much more often than in single patients [5]. It is noteworthy that the frequency of cerebral infarctions is determined, first of all, by the presence of type 2 diabetes and to a lesser extent depends on the severity of arterial hypertension. The frequency of detection of postischemic foci is higher in patients with diagnosed type 2 diabetes and insulin resistance [7]. At the same time, the point of view about the role of type 2 diabetes as an independent cause of cerebral infarction has not been confirmed in all studies [18].

Diffuse white matter lesion is leukoaraiosis, according to Hijdra et al. [3], significantly more often observed in patients with type 2 diabetes compared with individuals with normal carbohydrate metabolism. This dependence has not been confirmed in other studies [13].

In recent years, it has been established that type 2 diabetes is a risk factor not only for cerebral stroke, but also for chronic disorders of cerebral circulation, as well as processes accompanied by a progressive decline in cognitive functions. The results of population studies indicate a significant relationship between the presence of type 2 diabetes and the risk of both vascular dementia and Alzheimer's disease [4].

ISSN 27924025 (online), Published under Volume: 1 Issue: 4 in September2021

There is a certain genetic predisposition to the development of Alzheimer's disease in patients with type 2 diabetes. Thus, the combination of type 2 diabetes and the ApoE4 allele approximately doubles the risk of developing Alzheimer's disease compared to individuals with only the ApoE4 allele [35]. By itself, the presence of type 2 diabetes does not lead to the deposition of amyloid and the development of the clinical picture of Alzheimer's disease, but the available data indicate a significant increase in the number of neurofibrillary glomeruli and the accumulation of amyloid in individuals with this genotype, as well as the role of diabetes mellitus as a risk factor for Alzheimer's disease and mixed dementia [16]. However, not all studies support a role for type 2 diabetes as a risk factor for nonvascular dementia. Thus, a 5-year follow-up of 5574 individuals who initially had no cognitive impairment made it possible to establish that the existing type 2 diabetes was significantly associated with vascular dementia, as well as mild cognitive impairments that did not reach the degree of dementia. At the same time, no connection was established between disorders of carbohydrate metabolism and the development of Alzheimer's disease and the onset of mixed dementia [37]. Similar data on the relatively low risk of Alzheimer's disease in patients with type 2 diabetes were obtained as a result of 4-year follow-up of 1262 members of the multiethnic population of the elderly [18].

In patients with type 2 diabetes, an increase in the frequency and severity of cognitive impairments caused by vascular brain damage was found. The combination of type 2 diabetes and arterial hypertension is especially unfavorable. Interestingly, according to the results of the NHANES III study, in persons under the age of 60 who have not previously suffered a cerebral stroke, neither type 2 diabetes nor hypertension alone lead to significant impairments of higher cerebral functions. The combination of these two diseases is accompanied by a noticeable decrease in cognitive functions [9]. The mortality rate among these patients is 2.4 times higher than among persons of comparable age in the population.

Many large multicenter placebo-controlled studies have been devoted to the correction of hemostasis disorders for the purpose of secondary prevention of strokes and myocardial infarctions [11], which showed a greater efficacy of aspirin in combination with clopidogrel compared with aspirin monotherapy. The American Diabetes Association in 2001 [12] recommended the use of aspirin for primary prevention in diabetic patients with a high risk of macrovascular complications. However, acetylsalicylic acid blocks only one of the pathways of platelet activation and does not affect others, does not block the primary adhesion of platelets to the damaged endothelium, or the attachment of fibrinogen to its receptors on the surface. At the same time, it should be borne in mind that a significant part of patients (up to 30%) have low sensitivity to the drug or poorly tolerate it due to gastrointestinal complications, therefore, a consistently high level of platelet aggregation during treatment requires the simultaneous administration of another antiplatelet agent or a change preparation [13]. This determines the relevance of further search for antithrombotic drugs for primary prevention in patients with type 2 diabetes with a high cerebrovascular risk.

Conclusion. In conclusion, it can be stated that medicine has a wide range of knowledge about the risk factors for cerebrovascular diseases in type 2 diabetes and a wide diagnostic potential for their detection. Only a strategy aimed at maximizing correction of all existing risk factors can be recognized as effective.

Literature

1. Khodjiyeva D.T. Self-knowledge and self-affirmation are the key components of modern personality // european research: innovation in science, education and technology. - 2020 .- - S
2. Kario K., Matsuo T., Kobayashi H., Hoshide S., Shimada K. Hyperinsulinemia and hemostatic abnormalities are associated with silent lacunar cerebral infarcts in elderly hypertensive subjects. J. Am. Coll. Cardiol. 2001; 37:871-877.
3. Price T.R., Manolio T.A., Kronmal R.A., Kittner S.J., Yue N.C., Robbins J., Anton-Culver H., O'Leary DH. Silent brain infarction on magnetic resonance imaging and neurological abnormalities in community-dwelling older adults. The Cardiovascular Health Study CHS Collaborative Research Group. Stroke. 1997;28:1158-1164.
4. Howard G., Wagenknecht L.E., Cai J., Cooper L., Kraut M.A., Toole J.F. Cigarette smoking and other risk factors for silent cerebral infarction in the general population. Stroke. 1998;29:913- 917.
5. Longstreth W.T. Bernick C., Manolio T.A., Bryan N., Jungreis C.A., Price T.R. Lacunar infarcts defined by magnetic resonance imaging of 3660 elderly people: the Cardiovascular Health Study. Arch Neurol. 1998;55: 1217-1225.
6. Hijdra A., Verbeeten B., Verhulst J. Relation of leukoaraiosis to lesion type in stroke patients. Stroke 1990;21:890-894.

ISSN 27924025 (online), Published under Volume: 1 Issue: 4 in September2021

7. Henon H., Godefroy O., Lucas C., Pruvo J.P., Leys D. Risk factors and leukoaraiosis in stroke patients. Acta. Neurol. Scand. 1996 94(2):137-144.
8. Inzitari D., Diaz F., Fox A. Vascular risk factors and leukoaraiosis. Arch. Neurol. 1987;44:42-47.
9. Messier C. Diabetes, Alzheimer's disease and apolipoprotein genotype. Exp. Gerontol. 2003;38(9):941-6.
10. MacKnight C., Rockwood K., Awalt E., McDowell I. Diabetes mellitus and the risk of dementia, Alzheimer's disease and vascular cognitive impairment in the Canadian Study of Health and Aging. Dement. Geriatr. Cogn. Disord. 2002;14(2):77-83.
11. Xu W. L., Qiu C. X., Wahlin A., Winblad B., Fratiglioni L. Diabetes mellitus and risk of dementia in the Kungsholmen project. A 6-year follow-up study. Neurology 2004;63:1181-1186
12. Pavlik V. N., Hyman D. J., Doody R. Cardiovascular Risk Factors and Cognitive Function in Adults 30-59 Years of Age (NHANES III). Neuroepidemiology 2005;24:42-50.
13. Haan M.N., Wallace R. Can dementia be prevented? Brain aging in a population-based context. Annu. Rev. Public Health. 2004; 25:1-24.
14. Katakami N., Yamasaki Y., Hayaishi-Okano R., Ohtoshi K., Kaneto H., Matsuhisa M., Kosugi K., Hori M. Metformin or gliclazide, rather than glibenclamide, attenuate progression of carotid intima-media thickness in subjects with type 2 diabetes. Diabetologia. 2004;47(1 1):1906-1913.
15. Imomjonovich I. I. et al. Immunogenetic changes in kidney transplantation //International Engineering Journal For Research & Development. – 2021. – Т. 6. – №. 3. – С. 3-3.
16. Aronoff S, Rosenblatt S, Braithwaite S, et al. Pioglitazone hydrochloride monotherapy impruves glycemic control in the triatment of patients with type 2 diabetes. Diabetes Care.2000;23:1605-1611
17. Ходжиева Д. Т., Хайдарова Д. К., Хайдаров Н. К. Биофизические основы возникновения сирингомиелии: современные воззрения //Журнал неврологии и нейрохирургических исследований. – 2020. – Т. 1. – №. 3.
18. Ходжиева Д. Т. ВЛИЯНИЕ нейроиммунологических показателей больных с ишемическим инсультом на развитие постинсультной деменции //Новый день в медицине. – 2019. – №. 4. – С. 100-105.

ISSN 27924025 (online), Published under Volume: 1 Issue: 4 in September2021