УДК 616.89-005:614.1

O.A. Levada, A.S. Troyan

EPIDEMIOLOGICAL ASPECTS OF MAJOR VASCULAR NEUROCOGNITIVE DISORDER OVER THE LAST 10 YEARS: A BRIEF REVIEW

State Institution «Zaporizhzhia Medical Academy of Postgraduate Education Ministry of Health of Ukraine», Zaporizhzhia

olevada@zmapo.edu.ua

Background. According to DSM-5, major vascular neurocognitive disorder (VaMNCD) is the diagnostical construct that corresponds to the previously proposed term of vascular dementia in ICD-10 and DSM-IV classifications. Currently, VaMNCD is the second most common cause of major neurocognitive disorders (MNCDs) in elderly after Alzheimer's disease (MNCD-AD).

Objective. The aim was to synthesize evidence about some epidemiological aspects of VaMNCD over the past ten years.

Methods and materials. We searched PubMed and MEDLINE to find relevant data on VaMNCD.

Results and conclusions. According to clinical data, prevalence for VaMNCD among MNCDs is about 20%; the population prevalence for VaMNCD in the 65+ age group varies from 0.9 to 2%; with incidence rates between 0,75 and 11.4/1,000 person-years. The prevalence and incidence of VaMNCD increase with advancing age. According to autopsy data, the prevalence of «pure» VaMNCD ranges from 4 to 5,5%. The prevalence of mixed MNCD increases in older age cohorts. Vascular disorders most frequently linked with VaMNCD are atherosclerosis of cerebral arteries, small vessel disease, and cerebral amyloid angiopathy. Most common morphological types of VaMNCD are subcortical arteriosclerotic encephalopathy, multi-infarct encephalopathy, and strategic infarct dementia. Vascular, genetic, lifestyle risk factors, as well as the volume and location of the brain destruction, are the factors associated with VaMNCD. The presence of VaMNCD doubles the mortality of elderly persons. Economic burden of VaMNCD is higher than that of MNCD-AD. Donepezil treatment strategy is the most cost-effective in mild to moderate VaMNCD.

Key words:
major vascular neurocognitive disorder, prevalence, morphological forms, mortality, risk factors, economic burden.

Introduction. *Major vascular neurocognitive disorder* (VaMNCD) is a heterogeneous group of cognitive disorders associated with various types of cerebrovascular lesions. VaMNCD is a subtype of major neurocognitive disorders (MNCDs), which are characterized by the evidence of significant cognitive decline from a previous level of performance in one or more cognitive domains; this decline interferes with independence in everyday activities, do not occur exclusively in the context of a delirium, and is not better explained by another mental disorder [1]. Table 1 represents the DSM-5 diagnostic criteria for VaMNCD. According to DSM-5, VaMNCD corresponds to the condition referred to in DSM-IV and ICD-10 as vascular dementia [1].

In this review we analyzed recent data on epidemiological aspects of VaMNCD published in PubMed and MEDLINE over the past ten years.

Prevalence of VaMNCD according to clinical research

Prevalence analysis of VaMNCD in different geographical regions is shown in Table 2. At the present time, epidemiological studies of VaMNCD use such indicators: the proportion of VaMNCD among other MNCDs (represented as a percentage), the prevalence of VaMNCD in a separate age cohort (percentage), and incidence (a number of cases per 1,000 person-years). Prevalence indicates the total number of existing cases of a disease in a population at a given time. Incidence shows the number of new cases of a disease that develop in a given period in a defined population.

The majority of research show that the proportion of VaMNCD among other types of MNCDs in the older age groups is about 20% [2-4]. This rate is mostly the same in different geographic regions. According to this indicator, VaMNCD takes the second place in the group of MNCDs just after Alzheimer's disease. The proportion of VaMNCD in different geographical regions has aligned only in the last decade of study the disorder [5]. Moreover, the ratio of different MNCDs in Asian countries becomes similar to that of Western countries [6]. This change may be attributed to the unified approaches for the diagnosis of MNCDs (dementias), which are being implemented around the world.

Prevalence rates of VaMNCD for age groups of 65+ years ranges from 0.9 to 2.1%. This parameter has no significant associations with a geographical region. The incidence of VaMNCD varies from 0.75 to 11.4 cases per 1,000 person-years. The incidence is slightly lower in European countries compared with the United States or China.

Prevalence indicators of VaMNCD increase progressively with the age of observed people (Table 3). The incidence

Table 1. The diagnostic criteria for VaMNCD [1]

A	The criteria are met for major neurocognitive disorder.
B	The clinical features are consistent with a vascular etiology, as suggested by either of the following: 1. Onset of the cognitive deficits is temporally related to one or more cerebrovascular events. 2. Evidence for decline is prominent in complex attention (including processing speed) and frontal-executive function.
C	There is evidence of the presence of cerebrovascular disease from history, physical examination, and/or neuroimaging considered sufficient to account for the neurocognitive deficits.
D	The symptoms are not better explained by another brain disease or systemic disorder.
Probable vascular neurocognitive disorder is diagnosed if one of the following is present; otherwise possible vascular neurocognitive disorder should be diagnosed: 1. Clinical criteria are supported by neuroimaging evidence of significant parenchymal injury attributed to cerebrovascular disease (neuroimaging-supported). 2. The neurocognitive syndrome is temporally related to one or more documented cerebrovascular events. 3. Both clinical and genetic (e.g., cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy) evidence of cerebrovascular disease is present.	
Possible vascular neurocognitive disorder is diagnosed if the clinical criteria are met but neuroimaging is not available and the temporal relationship of the neurocognitive syndrome with one or more cerebrovascular events is not established.	
Coding note: For probable major vascular neurocognitive disorder, with behavioral disturbance, code 290.40 (F01.51). For probable major vascular neurocognitive disorder, without behavioral disturbance, code 290.40 (FOI .50). For possible major vascular neurocognitive disorder, with or without behavioral disturbance, code 331.9 (G31.9). An additional medical code for the cerebrovascular disease is not needed.	

Table 2. Prevalence and incidence of VaMNCD according to clinical research

Basic information about the research	Epidemiological indicators of VaMNCD		
	Proportion of VaMNCD among other MNCD, %	Prevalence, %	Incidence, index of cases per 1,000 person-years
EUROPEAN COUNTRIES			
Virués-Ortega et al. (2011) [4]: **Spain**; age 75+ years	18.6	1.4	–
Schrijvers et al. (2012) [10]:**Rotterdam** Study; 2000; age 60–90 years	–	–	0.75
Dimitrov et al. (2012) [11]: **Bulgaria**; age 65+ years	–	2.0	–
Imfeld et al. (2013) [12]: **the UK**; 1998 – 2008; age 65+ years	–	–	0.99
NORTH AMERICAN COUNTRIES			
Plassman et al. (2011) [7]: **the USA**; 2001-2009; age 72+ years	18.7	–	2.1 in cohort of 70-79 years; 11.4 in cohort of 80-89 years
SOUTH AMERICAN COUNTRIES			
Molero et al. (2007) [13]: **Venezuela**; age 55+ years	27.0	–	–
Kalaria et al. (2008) [14]: **Brazil**; 2002–2008; age 65+ years	–	0.9	–
Grinberg et al. (2013) [15]: **Brazil**; age 50+ years	21.2	–	–
ASIAN COUNTRIES			
Ikejima et al. (2012) [3]: **Japan**; 2009; age 65-99 years	18.9	–	–
Zhang et al. (2012) [16]: **China**; meta-analysis of 40 studies from 1980 to 2010; age 60+ years	–	0.9	–
Chan et al. (2013) [17]: **China**; a systematic review of research; 1990 – 2010; age 65-99 years	–	1.04	2.42
Wu et al. (2013) [18]: **China, Hong Kong, Taiwan**; meta-analyses of 76 studies between 1980–2012; age 60+ years	28.5	–	–
Cheng et al. (2014) [19]: **Shanghai**; age 60+ years	–	1.43	–
Jia et al. (2014) [20]: **China**; 2008-2009; age 65+ years	–	1.50	–
Ji et al. (2015) [21]: **China**; 2011-2012; age of 60+ years	–	1.7	–
Kim et al. (2011) [22]: **South Korea**; age 65+ years	–	2.0	–
Kim et al. (2014) [23]: **Korea**; a systematic review and meta-analyses of 704 studies; 1990–2013; age 65+ years	–	2.1	–
AFRICAN COUNTRIES			
El Tallawy et al. (2012) [2]: **Egypt**; age 50+ years	28.7	–	–
George-Carey et al. (2012) [24]: **Africa**; a systematic analysis; age 60+ years	26.9	1.0	–

of VaMNCD has the same pattern [7]. Men dominate significantly among patients at age 65-69 years, and women - in the age cohort 85+ years [8].

Some evidence suggests that the prevalence of VaMNCD was increasing during last decades. Sekita et al. (2010) studied the prevalence of dementia in a general Japanese population aged older 65 years and revealed that the prevalence of VaMNCD increased significantly over the last 10 years [9]. Thus, prevalence of VaMNCD in 1998 and 2005 had such indicators 1.5% and 2.5%, respectively. This trend requires further studying.

Table 3. VaMNCD prevalence in different age/gender groups (adapted from [8])

Age groups	65-69 years	90+ years
All MNCDs	0.8%	28.5%
VaMNCD	0.3%	5.2%
Age groups	65-69 years	85+ years
Men	0.5%	3.6%
Women	0.1%	5.8%

Prevalence of VaMNCD according to autopsy studies

Jellinger and Attems (2010) performed a retrospective study of prevalence of various MNCDs according to typical neuropathological changes in 1,110 consecutive autopsy cases of demented patients older 60+ years [25]. VaMNCD pathology was found to be in 10.8% of the total cohort, including mixed MNCD in 5.5%. Similar results were obtained in a British study of brain autopsy in 213 participants with sufficient information of MNCD diagnosis at the end of their lives: prevalence of «pure» VaMNCD was 4% [26]. It was highlighted that the prevalence of «pure» VaMNCD declined progressively from the age of 60 to 90+ years; meanwhile, the prevalence of mixed forms increased [25].

Vessel diseases causing VaMNCD

Vascular diseases such as atherosclerosis of cerebral arteries, cerebral small vessel disease, and cerebral amyloid angiopathy are the most frequent causes of VaMNCD [27-29]. These vascular lesions frequently occur in the elderly brain and their frequency and severity increase with advancing age [25]. Less common forms of vascular pathology to cause VaMNCD are different types of vasculitis and inherited diseases that affect vessel integrity, e.g., cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) [27].

Atherosclerosis is a degenerative disorder that affects large and medium-sized arteries; that results in proliferation of intima and accumulation of cholesterol within the vessel wall. These processes lead to the generation and calcification of atherosclerotic plaques, which may rupture with subsequent thrombosis. The thrombus can lead to the vessel occlusion or embolization and occlusion of a smaller distant artery [30].

Small vessel disease includes arteriosclerosis, lipohyalinosis, and arteriolosclerosis of small arteries caused by age, hypertension or genetic factors (e.g., CADASIL). These changes of the small vessel wall are similar to that of larger blood vessels except for calcifications. *Small vessel disease may lead to* lacunar infarcts, microinfarcts, hemorrhages, and microbleeds [30]. First, it affects arteries of the basal ganglia, afterward expands into the peripheral white matter, leptomeningeal arteries, thalamic and cerebellar white matter vessels; and finally involves brain stem arteries. As it was previously shown, cortical vessels are not usually involved in *small vessel disease (see review [27]).*

Sporadic forms of cerebral amyloid angiopathy are associated with the deposition of the amyloid β-protein in the cerebral and leptomeningeal vessel walls [28]. These deposits may cause wall ruptures of the affected vessel and lead to hemorrhage, microbleeds, capillary occlusion with blood flow disturbances, and consequently to microinfarcts [31]. The deposition of amyloid β-protein and other proteins, e.g., prion protein and cystatin C [32], characterize familial forms of cerebral amyloid angiopathy. *Cerebral amyloid angiopathy most* frequently starts in leptomeningeal and neocortical arteries, veins, capillaries in such cortical regions: hippocampus, entorhinal and cingulate cortex, amygdala. Meanwhile, blood vessels of the brainstem are involved at a later stage [28].

Pathomorphological types of VaMNCD

At the present day, the most prevalent pathomorphological types of VaMNCD are subcortical arteriosclerotic encephalopathy (most prevalent), multi-infarct encephalopathy, and strategic infarct type dementia [25]. Age or sex of the patients has no influence on the frequency of these lesions.

Pathomorphological signs of *subcortical arteriosclerotic encephalopathy* include the presence of lacunas, microinfarcts, and microbleeds that predominantly affect central white matter and subcortical structures (thalamus, basal nuclei, internal capsule, brainstem, white matter of the cerebellum). These lesions are caused by s*mall vessel disease, which was* described earlier [33].

Multi-infarct encephalopathy comprises large territorial infarcts due to large artery lesions of the brain, distal field infarcts in the areas of collateral blood flow of artery pools (basically associated with hemodynamic events and carotid artery stenosis), and embologenic microinfarcts in different cerebral areas [27].

Strategic infarct dementia is associated with unilateral lesions, which involve functionally important for cognitive processing brain areas and neuronal circuits such as the thalamus, medial temporal zone/hippocampus, frontal cortex, basal nuclei, etc. These lesions predominantly occur due to the affection of large brain arteries, embolic events, hemodynamic disturbances, and cerebral ischemia of diverse etiology [27, 29].

Risk factors of VaMNCD

Pathogenic risk factors linked with VaMNCD are conventionally subdivided into vascular, genetic, lifestyle [34], associated with the volume and location of brain destruction [27].

Valid studies found relationships between VaMNCD and hypertension [12], the level of carotid arteries atherosclerosis [35-37], hyperlipidemia [38], diabetes [39], and atrial fibrillation [12, 40].

Genetic factors may affect both the occurrence and the course of VaMNCD [27]. ApoE ε4 and ε2 may be involved in some microvascular changes of the brain in VaMNCD according to their amyloidogenic role. Notch3 mutation is responsible for the development of CADASIL, a hereditary form of the subcortical type of VaMNCD [41].

Lifestyle factors that increase the likelihood of the occurrence of VaMNCD are incorrect diet preferences [34], obesity, and physical inactivity [42].

Essential pathogenic factors of VaMNCD include *the volume, location, and amount of brain damage*. Brain tissue destruction greater than 100 ml, infarction in the dominant hemisphere, left angular gyrus, bilateral lesions in head of the caudate nuclei and other parts of basal ganglia, cortico-thalamic and thalamocortical pathways, and hippocampus significantly increase the risk of VaMNCD [27].

Special systematic review of 133 studies over a period from 1980 to 2011 [43] showed that low education also increased the risk of MNCDs incidence, including the vascular one. The authors proved that in individuals with higher education clinical features of VaMNCD occurred at more severe brain lesions, but later those patients demonstrated more rapid functional decline.

Mortality in VaMNCD

According to average data, VaMNCD doubles the mortality [17, 44]. The degree of VaMNCD correlates with the risk of mortality from mild (HR = 2.23; CI: 1.77-2.82) to moderate (HR = 3.10; CI: 2.47-3.89), and severe VaMNCD (HR = 4.98; CI: 3.85-6.44) [45]. Factors associated with higher mortality are senior age [45, 46], male gender, comorbidity [45], small brain weight, more significant microvascular lesions of subcortical brain regions, and multiple vascular pathologies [46]. Although, age remains the main risk factor for mortality in VaMNCD patients [47].

Strand et al. (2013) studied midlife vascular risk factors associated with mortality due to different forms of MNCDs in old and oldest-old age groups [48]. The researchers found that significant midlife factors that increased death from VaMNCD were high cholesterol levels (>7.80 mmol/l), diabetes, and low body mass index (<20 kg/m^2 vs. 20-25 kg/m^2). Arterial hypertension and smoking tobacco in the middle age displayed no significant association with the risk of MNCD's death in older age periods of life.

The burden of VaMNCD

Currently, VaMNCD is one of the diseases to cause the most negative impact on health expenditures [49]. According to some reports, patients with VaMNCD require higher costs compared with Alzheimer's disease patients [50].

The Spanish National Health Institute reported that the average annual cost of treatment per inpatient (direct costs) with VaMNCD was ~$22 631; this value was significantly higher than per-patient costs during mild vascular neurocognitive disorder (mild vascular cognitive impairment according to DSM-IV) [49]. In Argentina, the annual direct cost for treatment one patient with VaMNCD is $5 112 [51]. The proportion of hospitalization costs dominates among direct costs of VaMNCD. This rate is greater for VaMNCD compared with Alzheimer's disease and frontotemporal dementia, in which the majority of spending goes on anti-dement and antipsychotic drugs. Among different types of VaMNCD, the expenditures of patients with Binswanger's disease (microvascular subcortical leukoencephalopathy) are the highest [49].

In a systematic review, Wong et al. (2009) studied the cost-effectiveness of donepezil, galantamine, rivastigmine, and memantine treatment in standard doses in mild to moderate stages of VaMNCD [52]. Treatment with cholinesterase inhibitors or memantine were more effective, but also more expensive than standard care for VaMNCD patient. According to cost-effectiveness, the donepezil strategy (10 mg per day) dominated the alternatives. The other treatment strategies were less effective and more costly.

Separate studies analyzed the burden of VaMNCD for persons that surround patients and take care of them. It was proven that psychotic symptoms and impaired activity of everyday living in VaMNCD patients cause more significant caregiver burden than cognitive dysfunction itself [53].

Conclusion

At the present time, VaMNCD is the second most common cause of MNCDs in elderly persons after MNCD due to Alzheimer's disease. Prevalence for VaMNCD among MNCDs is about 20%, the population prevalence for VaMNCD varies from 0.9 to 2% in the age group older 65 years; incidence varies from 0.75 to 11.4/1,000 person-years, increasing with age. Most common morphological types of VaMNCD are subcortical arteriosclerotic encephalopathy, multi-infarct encephalopathy, and strategic infarct dementia. Vascular, genetic, lifestyle risk factors, as well as the volume and location of the brain destruction, are the factors associated with VaMNCD. The presence of VaMNCD increases the mortality of elderly persons about two times. The donepezil treatment strategy is the most cost-effective in mild to moderate VaMNCD.

Список використаної літератури / References

1. *American Psychiatric Association: Diagnostic and Statistical manual of mental Disorders. American Psychiatric Publishing, Washington, DC 2013, 5th ed., 591-645.*
2. *El Tallawy HN, Farghly WM, Shehata GA et al: Prevalence of dementia in Al Kharga District, New Valley Governorate, Egypt. Neuroepidemiology 2012, 38, 130–137.*
3. *Ikejima C, Hisanaga A, Meguro K et al: Multicentre population-based dementia prevalence survey in Japan: a preliminary report. Psychogeriatrics 2012, 12, 120–123.*
4. *Virués-Ortega J, de Pedro-Cuesta J, Vega S et al: Prevalence and European comparison of dementia in a ≥75-year-old composite population in Spain. Acta Neurol Scand 2011, 123, 316–324.*
5. *Asada T: Prevalence of dementia in Japan: past, present and future. Rinsho Shinkeigaku 2012, 52, 962–964.*
6. *Catindig JA, Venketasubramanian N, Ikram MK, Chen C: Epidemiology of dementia in Asia: insights on prevalence, trends and novel risk factors. Neurol Sci 2012, 321, 11–16.*
7. *Plassman BL, Langa KM, McCammon RJ et al: Incidence of dementia and cognitive impairment, not dementia in the United States. Ann Neurol 2011, 70, 418–426.*
8. *Jellinger KA: Morphologic diagnosis of «vascular dementia» – a critical update. J Neurol Sci 2008, 270, 1–12.*
9. *Sekita A, Ninomiya T, Tanizaki Y et al: Trends in prevalence of Alzheimer's disease and vascular dementia in a Japanese community: the Hisayama Study. Acta Psychiatr Scand 2010, 122, 319–325.*
10. *Schrijvers EM, Verhaaren BF, Koudstaal PJ et al: Is dementia incidence declining?: Trends in dementia incidence since 1990 in the Rotterdam Study. Neurology 2012, 78, 1456–1463.*
11. *Dimitrov I, Tzourio C, Milanov I et al: Prevalence of dementia and mild cognitive impairment in a Bulgarian urban population. Am J Alzheimers Dis Other Demen 2012, 27, 131–135.*
12. *Imfeld P, Brauchli Pernus YB et al: Epidemiology, co-morbidities, and medication use of Patients with Alzheimer's disease or vascular dementia in the UK. J Alzheimers Dis 2013, 35, 565–573.*
13. *Molero AE, Pino-Ramírez G, Maestre GE: High prevalence of dementia in a Caribbean population. Neuroepidemiology 2007, 29, 107-12.*
14. *Kalaria RN, Maestre GE, Arizaga R et al. for the World Federation of Neurology Dementia Research Group: Alzheimer's disease and vascular dementia in developing countries: prevalence, management, and risk factors.* Lancet Neurol 2008, 7, 812–826.
15. *Grinberg LT, Nitrini R, Suemoto CK et al: Prevalence of* dementia *subtypes in a developing country: a clinicopathological study. Clinics (Sao Paulo) 2013, 68, 1140-1150.*
16. *Zhang Y, Xu Y, Nie H et al: Prevalence of dementia and major dementia subtypes in the Chinese populations: a meta-analysis of dementia prevalence surveys, 1980-2010. J Clin Neurosci 2012, 19, 1333–1337.*

17. *Chan KY, Wang W, Wu JJ et al: Epidemiology of Alzheimer's disease and other forms of dementia in China, 1990-2010: a systematic review and analysis. Lancet 2013, 381, 2016–2023.*
18. *Wu Y, Lee HY, Norton S et al: Prevalence studies of dementia in mainland China, Hong Kong and Taiwan: a systematic review and meta-analysis. PLoS One 2013, 8(6):e66252.*
19. *Cheng Q, Sun HX, Ye FL et al: Dementia among elderly in Shanghai suburb: a rural community survey. J Alzheimers Dis 2014, 39, 883-9.*
20. *Jia J, Wang F, Wei C, Zhou A et al: The prevalence of dementia in urban and rural areas of China. Alzheimers Dement 2014, 10, 1-9.*
21. *Ji Y, Shi Z, Zhang Y et al: Prevalence of dementia and main subtypes in rural northern China. Dement Geriatr Cogn Disord 2015, 39, 294-302.*
22. *Kim KW, Park JH, Kim MH et al: A nationwide survey on the prevalence of dementia and mild cognitive impairment in South Korea. J Alzheimers Dis 2011, 23, 281–291.*
23. *Kim YJ, Han JW, So YS et al: Prevalence and trends of dementia in Korea: a systematic review and meta-analysis. J Korean Med Sci 2014, 29, 903-12.*
24. *George-Carey R, Adeloye D, Chan KY et al: An estimate of the prevalence of dementia in Africa: A systematic analysis.* J Glob Health 2012, 2, 020401.
25. *Jellinger KA, Attems J: Prevalence of dementia disorders in the oldest-old: an autopsy study. Acta Neuropathol 2010, 119, 421–433.*
26. *Brayne C, Richardson K, Matthews FE et al: Neuropathological correlates of dementia in over-80-year-old brain donors from the population-based Cambridge city over-75s cohort (CC75C) study. J Alzheimers Dis 2009, 18, 645–658.*
27. *Jellinger KA: Pathology and pathogenesis of vascular cognitive impairment - a critical update. Front Aging Neurosci 2013, 5, 17.*
28. *Attems J, Jellinger K, Thal DR, Van Nostrand W: Review: sporadic cerebral amyloid angiopathy. Neuropathol Appl Neurobiol 2011, 37, 75–93.*
29. *Ferrer I: Cognitive impairment of vascular origin: neuropathology of cognitive impairment of vascular origin. J Neurol Sci 2010, 15, 139–149.*
30. *Grinberg LT, Thal DR: Vascular pathology in the aged human brain. Acta Neuropathol 2010, 119, 277–290.*
31. *Okamoto Y, Yamamoto T, Kalaria RN et al: Cerebral hypoperfusion accelerates cerebral amyloid angiopathy and promotes cortical microinfarcts. Acta Neuropathol 2012, 123, 381–394.*
32. *Revesz T, Holton JL, Lashley T et al: Genetics and molecular pathogenesis of sporadic and hereditary cerebral amyloid angiopathies. Acta Neuropathol 2009, 118, 115–130.*
33. *Rosenberg GA: Neurological diseases in relation to the blood-brain barrier. J Cereb Blood Flow Metab 2012, 32, 1139–1151.*
34. *Akinyemi R., Mukaetova-Ladinska EB, Attems J et al: Vascular risk factors and neurodegeneration in ageing related dementias: Alzheimer's disease and vascular dementia. Curr Alzheimer Res 2013, 10, 642-653.*
35. *Arntzen KA, Mathiesen EB: Subclinical carotid atherosclerosis and cognitive function. Acta Neurol Scand Suppl 2011, 191, 18–22.*
36. *Viticchi G, Falsetti L, Vernieri F et al: Vascular predictors of cognitive decline in patients with mild cognitive impairment. Neurobiol Aging 2012, 33, 1127 .e1-9.*
37. *Wendell CR, Waldstein SR, Ferrucci L et al: Carotid atherosclerosis and prospective risk of dementia. Stroke 2012, 43, 3319-3324.*
38. *Solomon A, Kivipelto M, Wolozin B et al: Midlife serum cholesterol and increased risk of Alzheimer's and vascular dementia three decades later. Dement Geriatr Cogn Disord 2009, 28, 75–80.*
39. *Ahtiluoto S, Polvikoski T, Peltonen M et al: Diabetes, Alzheimer disease, and vascular dementia: a population-based neuropathologic study. Neurology 2010, 75, 1195–11202.*
40. *Puccio D, Novo G, Baiamonte V et al: Atrial fibrillation and mild cognitive impairment: what correlation? Minerva Cardioangiol 2009, 57, 143–150.*
41. *Chabriat H, Joutel A, Dichgans M et al: Cadasil. Lancet Neurol 2009, 8, 643–653.*
42. *Staekenborg SS, van Straaten EC, van der Flier WM et al: Small vessel versus large vessel vascular dementia: risk factors and MRI findings. J Neurol 2008, 255, 1644–1651; discussion 1813–1814.*
43. *Meng X, D'Arcy C: Education and dementia in the context of the cognitive reserve hypothesis: a systematic review with meta-analyses and qualitative analyses. PLoS One 2012, 7, e38268.*
44. *Wu XG, Tang Z, Fang XH et al: A cohort study on mortality and risk of death among population diagnosis as dementia, at base-line survey, in Beijing. Zhonghua Liu Xing Bing Xue Za Zhi 2011, 32, 973–977.*
45. *Villarejo A, Benito-León J, Trincado R et al: Dementia-associated mortality at thirteen years in the NEDICES Cohort Study. J Alzheimers Dis 2011, 26, 543–551.*
46. *Matthews FE, Brayne C, Lowe J et al: Epidemiological pathology of dementia: attributable-risks at death in the Medical Research Council Cognitive Function and Ageing Study. PLoS Med 2009, 6, e1000180. d.*
47. *Nilsson K, Gustafson L, Hultberg B: Survival in a large elderly population of patients with dementia and other forms of psychogeriatric diseases. Dement Geriatr Cogn Disord 2011, 32, 342–350.*
48. *Strand BH, Langballe EM, Hjellvik V et al: Midlife vascular risk factors and their association with dementia deaths: results from a Norwegian prospective study followed up for 35 years. J Neurol Sci 2013, 324, 124–130.*
49. *Ramos-Estebanez C, Moral-Arce I, Rojo F et al: Vascular cognitive impairment and dementia expenditures: 7-year inpatient cost description in community dwellers. Postgrad Med 2012, 124, 91–100.*
50. *Shinagawa S, Nakamura S, Iwamoto M et al: Longitudinal changes in the government-certified index stage and requisite costs for long-term care insurance system among the community-dwelling demented elderly in Japan. Int J Alzheimers Dis 2013, 2013:164919.*
51. *Rojas G, Bartoloni L, Dillon C et al: Clinical and economic characteristics associated with direct costs of Alzheimer's, frontotemporal and vascular dementia in Argentina. Int Psychogeriatr 2011, 23, 554-561.*
52. *Wong CL, Bansback N, Lee PE, Anis AH: Cost-effectiveness: cholinesterase inhibitors and memantine in vascular dementia. Can J Neurol Sci 2009, 36, 735–739.*
53. *Yeager CA, Hyer LA, Hobbs B, Coyne AC: Alzheimer's disease and vascular dementia: the complex relationship between diagnosis and caregiver burden. Issues Ment Health Nurs 2010, 31, 376–384.*

ЭПИДЕМИОЛОГИЧЕСКИЕ АСПЕКТЫ БОЛЬШОГО СОСУДИСТОГО НЕЙРОКОГНИТИВНОГО РАССТРОЙСТВА ЗА ПОСЛЕДНИЕ ДЕСЯТЬ ЛЕТ: КРАТКИЙ ОБЗОР

О.А. Левада, А.С. Троян

Актуальность. Согласно пятому изданию Диагностического и статистического руководства по психическим расстройствам (DSM-5), большое сосудистое нейрокогнитивное расстройство (БСНР) соответствует ранее предложенному термину сосудистой деменции в классификациях МКБ-10 и DSM-IV. На современном этапе БСНР является вторым по распространённости большим нейрокогнитивным расстройством (БНР) среди лиц старших возрастных групп после БНР вследствие болезни Альцгеймера (БНР-БА).

Цель. Обобщить сведения о некоторых эпидемиологических аспектах БСНР за последние десять лет.

Материалы и методы. Поиск соответствующей информации о БСНР проведён с помощью баз данных PubMed и MEDLINE.

Результаты и выводы. По данным клинических исследований, в возрастной когорте 65 и больше лет его удельный вес составляет около 20%, распространённость – от 0,9 до 2%, заболеваемость – 0,75 – 11,4 случаев на 1000 персоно-лет. Показатели распространённости и заболеваемости растут с увеличением возраста. По данным патологоанатомических исследований, частота «чистых» БСНР находится в пределах от 4 до 5,5%. По мере старения возрастает количество смешанных форм БНР. Наиболее распространённой сосудистой патологией, приводящей к БСНР, является атеросклероз церебральных артерий, болезнь мелких сосудов головного мозга и церебральная амилоидная ангиопатия. Преобладающими морфологическими типами БСНР являются субкортикальная энцефалопатия вследствие артериолосклероза, мультиинфарктная деменция и деменция вследствие инфаркта в стратегически важных зонах. Факторы риска возникновения БСНР разделяют на сосудистые, генетические, связанные с объёмом и локализацией поражения головного мозга и вызванные стилем жизни. БСНР увеличивает риск смерти лиц пожилого возраста приблизительно в 2 раза. Прямые затраты на пациента с БСНР превышают таковые на пациента с БНР-БА. По показателю «затраты – эффективность» назначение донепезила при лёгких и умеренных формах БСНР является наиболее целесообразным.

Ключевые слова: большое сосудистое нейрокогнитивное расстройство, распространённость, морфологические формы, смертность, факторы риска, экономические затраты.

ЕПІДЕМІОЛОГІЧНІ АСПЕКТИ ВЕЛИКОГО СУДИННОГО НЕЙРОКОГНІТИВНОГО РОЗЛАДУ ЗА ОСТАННІ ДЕСЯТЬ РОКІВ: СТИСЛИЙ ОГЛЯД

О.А. Левада, О.С. Троян

Актуальність. Згідно з п'ятим виданням Діагностичного та статистичного керівництва з психічних розладів (DSM-5), великий судинний нейрокогнітивний розлад (ВСНР) відповідає терміну судинна деменція, зазначеному у класифікаціях МКХ-10 та DSM-IV. На сучасному етапі ВСНР є другим за поширеністю великим нейрокогнітивним розладом (ВНР) серед осіб старших вікових груп, одразу після ВНР внаслідок хвороби Альцгеймера (ВНР-ХА).

Мета. Узагальнити дані про деякі епідеміологічні аспекти БСНР за останні десять років.

Матеріали та методи. Пошук релевантної інформації стосовно ВСНР виконано за допомогою баз даних PubMed та MEDLINE.

Результати та висновки. За даними клінічних досліджень, у віковій когорті 65 і більше років його питома частка складає близько 20%, поширеність – від 0,9 до 2%, захворюваність – 0,75 – 11,4 випадків на 1000 персоно-років. Показники поширеності та захворюваності ростуть зі збільшенням віку. За даними патологоанатомічних досліджень, частота «чистих» форм ВСНР коливається в межах від 4 до 5,5%. Зі збільшенням віку зростає кількість змішаних форм ВНР. Найпоширенішою судинною патологією, що призводить до розвитку ВСНР, є атеросклероз церебральних артерій, хвороба дрібних судин головного мозку та церебральна амілоїдна ангіопатія. Переважаючими морфологічними типами ВСНР є субкортикальна енцефалопатія внаслідок артеріолосклерозу, мультиінфарктна деменція та деменція внаслідок інфаркту в стратегічно важливих зонах. Фактори ризику виникнення ВСНР поділяють на судинні, генетичні, пов'язані з об'ємом і локалізацією ураження головного мозку, а також спричинені стилем життя. ВСНР збільшує ризик смертності серед осіб похилого віку вдвічі. Безпосередні витрати на пацієнта з ВСНР перевищують ті, що йдуть на хворого з ВНР-ХА. За показником «витрати – ефективність» призначення донепезилу при легких і помірних формах ВСНР є найдоцільнішим.

Ключові слова: великий судинний нейрокогнітивний розлад, поширеність, морфологічні форми, смертність, фактори ризику, економічні витрати.