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MEETING REPORT

NEOTRIC ADVANCES IN VASCULAR DEMENTIA

HIGHLIGHTS FROM THE 7TH INTERNATIONAL CONGRESS ON VASCULAR DEMENTIA, OCTOBER 20-23, 2011, RIGA, LATVIA

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SUMMARY

Dementia is a leading cause of memory impairment and disability. The two main types of dementia are primary degenerative dementia (e.g., Alzheimer´s disease) and vascular dementia. Vascular dementia is a group of disorders due to cerebrovascular disease of various etiologies, mechanisms and evaluations. The 7th International Congress on Vascular Dementia focused on new developments in the area of dementia, with special attention to cerebrovascular diseases affecting cognition in different fields, to deliberate on large- and small-vessel brain diseases and how they contribute to cognitive decline. The congress concentrated mainly on Alzheimer's disease and attempted to identify specific biological and psychological markers, if any, for vascular dementia, as well as genetic factors. The 8th International Congress on Vascular Dementia will be held in Athens, Greece, in 2013 and will be an excellent platform to discuss new strategies and tools that can connect and engage scientific and public conversation to advance knowledge, learning and engagement on vascular dementia.

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INTRODUCTION

The 7th International Congress on Vascular Dementia held in Riga, Latvia, October 20-23, 2011, attracted more than 500 participants, including neuroscientists from all over the world, and continued for 4 days, which involved plenary sessions, a Nycomed satellite symposium, poster presentations and other opportunities to discuss the challenges associated with the diagnosis and management of patients with vascular dementia or Alzheimer's disease with cerebrovascular disease. This congress is an annual, worldwide event reflecting its core mission of promoting international collaboration and the exchange of scientific information. Recent topics discussed at the meeting were the global economic cost of dementia, diagnostic and biomarkers in vascular cognitive impairment, post-stroke cognitive impairment, vascular depression and neuroregeneration in neurodegenerative disorders (see Table I).

GLOBAL ECONOMIC COST OF DEMENTIA

Dementia is affecting every healthcare system in the world, and large amounts are spent on caring for people with dementia. A proper understanding of the societal costs of dementia and how this impacts upon families, health and social care services and governments is fundamental for better policies at the international and national level to improve the lives of people with dementia. Alzheimer's Disease International launched the World Alzheimer Report 2009 with a systematic review of prevalence studies worldwide. The 2010 report was based on these numbers for a societal cost of illness study. Annual costs per person with dementia for each country have been applied to the estimated number affected in that country, and then aggregated up to the level of World Health Organization regions and World Bank income groupings. The total estimated worldwide costs of dementia were USD 604 billion in 2010. About 70% of the costs occur in Western Europe and North

Table I. *Overview of experimental studies.*

Ref.		
8.	Objective	This study aimed to examine the effects of two strong statins (atorvastatin 30 mg/kg/day and pitavastatin 3 mg/kg/day p.o.) on senile plaque (SP) and inflammatory responses in the brains of an amyloid precursor protein (APP) transgenic (Tg) mouse model of Alzheimer's disease (AD) from 5 to 20 months of age
	Outcomes	Results of immnohistological analysis showed that the sum of each SP size progressively increased with age until 20 months in the APPTg mice, and the numbers of monocyte chemotactic protein 1 (Mcp-1)-positive neurons. In the APPTg mice treated with the statins, the number of Mcp-1-positive neurons was first reduced as early as at 10 months; then the number of lupus brain antigen 1 (Lba-1)-positive microglias began to show a reduction at 15 months, that of TNF-α-positive neurons at 15-20 months, and finally the sum of each SP size at 15-20 months, and it was suggested that the protective effect of these statins took 5 months in the present AD mouse model, that Mcp-1, Lba-1 and TNF-α and both statins have high potential as a preventive approach for patients at risk of AD
9.	Objective	This study aimed for clinical epidemiological investigation of neurodegenerative diseases. An initial multicenter-based descriptive study to assess the magnitude of neurodegenerative diseases, followed by a case-control study, was conducted in collaboration with other higher educational institutions. Data were collected from 197 subjects initially for the descriptive phase: 63 dementia cases and 103 age-matched controls for the analytic phase using a questionnaire for behavioral factors and genetic factors. The standard protocols were followed to measure the selected biochemical factors
	Outcomes	Results showed that dementia was the leading disease (72.1%) among the identified neurodegenerative diseases. In investigation of genetic determinants, as in other areas of inquiry, one-fifth (19.7%) of the identified cases revealed a positive family history. Among the known co-morbidities, type 2 diabetes, hyperlipidemia and hypertension were remarkable. The evidence of high percentages of patients suffering from different co-morbidities in combination suggested the potential scope for further investigations of biochemical markers such as glycemic status and serum adiponectin levels to explore the respective etiological role. Furthermore, a behavioral determinant, cigarette smoking, may be an important determinant for neurodegenerative diseases. It was concluded that the proportion of dementia cases was alarmingly high and type 2 diabetes was the most common co-morbidity
10.	Objective	This study aimed at assessing vascular lesions in brain tissue and was carried out by BrainNetEurope, including 36 neuropathologists. All participating neuropathologists work daily with diagnostics of post-mortem brain tissue obtained from subjects with various neurological conditions
	Outcomes	The results explain the various incidences (2-12%) for pure vascular dementia. Several neuropathological classifications/ definitions of vascular dementia have been suggested based on size of tissue loss, localization of tissue damage, large or small vessel disease, etc. It was concluded that consensus criteria for assessment and interpretation of vascular lesions, as well as a neuropathological definition of vascular dementia, are urgently needed
11.	Objective	The purpose of this study was to present a case study of a young patient who sustained a severe hemorrhagic brain injury and who subsequently experienced cognitive recovery of an unexpected degree
	Outcomes	This study concluded that psychopharmacological intervention played a significant role in the patient's cognitive and psychological recovery
12.	Objective	This study aimed to explore the risk factors and controlled targeted physical therapy to address specific physical impairments for rehabilitation of vascular dementia
	Outcomes	This study concluded that advances in traditional rehabilitation approaches for stroke patients, as well as other innovative approaches such as dual-tasking, constraint-induced therapy, bilateral upper extremity training and cognitive remediation, improve mobility
13.	Objective	This study assessed if a "continuum" between "pure" AD and vascular dementia also occurs in early-onset dementia (EOD). In 68 EOD patients followed at the Memory Clinic of Cardarelli Hospital, vascular risk factors and vascular brain burden were assessed
	Outcomes	Study results showed that in EOD a clear border between degenerative and vascular processes is unlikely and a continuum seems to exist. Attention to vascular burden may be useful in EOD similar to in late-onset dementia
14.	Objective	This study aimed to explore the mechanisms involved in neurodegeneration
	Outcomes	This study concluded that neurodegeneration is the cause of over 80% of cases of cognitive impairment and dementia. Neurodegeneration is the result of a cascade of pathogenetic processes that can be started in different and varied ways, such as alterations in protein structure, aggregation, deposition or clearance, synaptic failure, neurotransmitter deficits, mitochondrial dysfunction, oxidative stress, deficits in axonal transport, alteration in neurotrophic factors or their receptors, etc.
15.	Objective	This study tested the post-mortem hippocampi from AD and control cases for basal and insulin-stimulated phosphorylation levels of multiple proteins that comprise an important insulin signaling pathway known to be abnormal in type 2 diabetes
	Outcomes	It was concluded that there are negative relationships between episodic memory scores and basal levels of both activated IRS-1 kinases and their IRS-1 pS products. These and related findings indicate that the hippocampus in AD is insulin-resistant, that such resistance is closely associated with dysregulation of IRS-1, which may be due to chronic activation of multiple IRS-1 kinases, and that factors promoting brain insulin resistance are strongly associated with episodic memory impairments

Continued

Table I. *(Cont.) Overview of experimental studies.*

Ref.		
16.	Objective	The purpose of the study was to determine the features of cognitive disorders in patients with ischemic and hemorrhagic strokes of mild or moderate severity. The study included 17 patients, 6 of whom had hemorrhagic stroke and 11 ischemic stroke. The average age of the patients was 65 and 64 years for hemorrhagic and ischemic stroke, respectively. Patients in both groups were comparable in terms of existing risk factors
	Outcomes	It was concluded that cognitive disorders of varying severity occurred in all patients, but more pronounced changes were observed in patients with hemorrhagic stroke
17.	Objective	This study aimed to explore the clinical cardiovascular risk factors and neurobehavioral phenotypes that are mimicked in a hypertensive rat vascular cognitive impairment (VCI) model Clinical: general linear models investigated the association between predictors (e.g., carotid intima-media thickness, blood pressure) and cognitive outcomes (Stroop and symbol digit score). Preclinical: carotid artery stenosis surgery (i.e., guided by reduced forebrain perfusion) or sham surgery was performed in hypertensive rats. A series of neurological (gait-balance) and cognitive (active place and passive avoidance) tests were performed
	Outcomes	It was concluded that clinical hypertension and carotid stenosis were associated with Stroop interference score in a large (1,363) cohort. Preclinical: stenosis in hypertensive rats results in cognitive-behavioral changes that mimic VCI. Active place avoidance requires complex cognitive control and provides a sensitive indice of cognitive deficits (e.g., mimics executive function). This model combines cerebrovascular factors that produce VCI and provides a tool to probe VCI pathophysiology, mechanisms and interventions
18.	Objective	This study aimed to evaluate the association between inflammatory markers, hippocampal volume and long-term cognitive outcome in stroke patients. A total of 296 patients were included. Inflammatory markers, including C-reactive protein, white blood cell count, erythrocyte sedimentation rate (ESR), cognitive scores and magnetic resonance (MR) scan results (N = 164) were analyzed
	Outcomes	Results showed that inflammatory markers on admission were inversely correlated with cognitive scores at baseline, as well as 6, 12 and 18 months thereafter (i.e., $P = 0.016$ for ESR and 6-month memory score), and it was concluded that there was a strong relationship between peripheral inflammatory biomarkers and hippocampal volume, as well as cognitive performance, among post-stroke patients. This suggests that peripheral low-grade inflammation could relate to cognitive decline via hippocampal pathways
19.	Objective	This study aimed to describe and report the symptoms of dementia caused by brain arteriovenous malformation. MRI, color Doppler ultrasound, analysis of EEG and BEAM and DSA angiography were analyzed
	Outcomes	It was concluded that dementia caused by brain arteriovenous malformations is rare
20.	Objective	This study investigated the relationship between reactive oxygen species (ROS) with outcome and volume of the diffusion-weighted image (DWI) lesion after acute cerebrovascular stroke
	Outcomes	It was concluded that the production of ROS after cerebrovascular stroke may play a role as a mediator of lesion enlargement in cerebral ischemia. Plasma $ONOO^-$ concentration is an independent predictive factor for lesion enlargement in the acute phase of cerebrovascular stroke
21.	Objective	This study aimed to access the behavioral and psychological symptoms of dementia (BPSD) in patients with AD and vascular dementia. There were a total of 124 cases of AD and 122 of vascular dementia
	Outcomes	Study results showed that the profile of BPSD is different in AD and vascular dementia. In vascular dementia, BPSD may be the predominant presenting feature. BPSD, being potentially remediable, deserves due attention
22.	Objective	This study investigated the concordance between clinical and neuropathological diagnoses in neurodegenerative disorders. The study included 200 patients who had donated their brains to the Brain Bank of Navarra (Pamplona, Spain). All patients had been diagnosed with a neurodegenerative disorder during their lifetime (clinical diagnosis) and post-mortem (neuropathological diagnosis)
	Outcomes	The results of this study indicated that it is not entirely accurate to distinguish subtypes of dementia by the most obvious neuropathological alteration, since the underlying pathology often differs from the neuropathology implied by the dementia subtype. Hence, a "multidimensional approach" was suggested, whereby dementia syndromes are not categorized into isolated subtypes, but where dementia is treated as a multidimensional disease with syndromes characterized by a spectrum of neuropathological and clinical alterations
23.	Objective	This study aimed to elucidate the possible cellular mechanism of the neuroprotective pharmacological effects of Actovegin®, and investigated its effects on apoptosis and oxidative stress in vitro in rat primary neurons. Primary neurons were cultured for up to 10 days in the presence of increasing doses of actovegin (0.3-1000 mg/L). Total cell number, dendrite length and the number of excitatory synapses, i.e., the amount of the synaptic V-Glut1 protein, were measured by immunocytochemistry, followed by fluorescence microscopy. The apoptotic level in neurons after induction of apoptosis by beta-amyloid peptide $A\beta_{25-35}$ was assessed by the caspase-3 level
	Outcomes	Results showed that Actovegin® treatment yielded significantly increased maintenance of neuronal cells and total number of synapses, and could lower the level of activated caspase-3 in a concentration-dependent manner. In addition, Actovegin® reduced the cellular level of ROS in cultured neurons and it was concluded that the cellular effects observed suggest neuroprotective and antioxidant effects for Actovegin®, which could at least partially explain its therapeutic benefits

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Table I. *(Cont.) Overview of experimental studies.*

Ref.		
24.	Objective	This study aimed to investigate the association between a clinical diagnosis of probable AD (PAD) and incident dementia risk in people with diabetes
	Outcomes	It was concluded that elderly diabetic patients with PAD have a large increase in the risk of incident dementia, independent of sociodemographic factors, hypertension and glycosylated hemoglobin. This association reflects the potential role of vascular injury in the pathway between diabetes and increased dementia risk
25.	Objective	This study aimed to investigate the associations between the presence of cardiovascular risk factors in patients with mild cognitive impairment and an increased risk of progression to dementia, explore the effect of Mildronate® use, taking into account its antioxidant, neuroprotective, heart output-increasing and cerebral perfusion-improving properties; 120 patients (72 female, 48 male; average age 71.6 years) with mild cognitive impairment received Mildronate® 1000 mg daily for 2 months. The patients were closely followed clinically, with repeated neuropsychological assessment
	Outcomes	Results showed that 46 patients showed stable improvement in cognitive performance measures and in daily life. Forty patients trend back to baseline by 6-12 months; 34 patients had no improvement after treatment. There were no cases of worsening disease symptoms, confusion, hallucinations and psychosis. It was concluded that mild cognitive impairment can be preventable and sometimes reversible, although the evidence for this is not as complete as it is for the prevention of stroke. However, prompt treatment has been shown to help slow the effects of some dementias. The use of Mildronate® is an alternative to the cholinesterase inhibitors, especially in old patients, since Mildronate® has fewer side effects and the risks are minimal. This fact is crucial for patients aged up to 65 years
26.	Objective	This study involved a large number of clinically and pathologically well-characterized subjects sourced from a population-based series to test the hypothesis that the threshold for cognitive decline in AD varies among three more or less genetically defined ethnic groups: Africans, Caucasians and a mixed African-Caucasian group
	Outcomes	It was concluded that ethnic-specific genetic variations may have a direct impact on the cognitive reserve. Unraveling the genetic differences between Caucasians and Africans may lead to a better understanding about protective factors against AD-related neuropathological changes
27.	Objective	This study aimed to examine the association between after stroke depression (ASD) and quality of life (QOL) in people with stroke. A total of 40 participants (21 men and 19 women) with a mean age of 61.5 years (SD ± 3.5 years) were evaluated at an average of 8.7 ± 3.5 months after stroke. The relationship between ASD and QOL was examined in a cross-sectional study in the subjects more than 6 months after stroke. Participants were those who attended the outpatient clinics of two rehabilitation centers in Tehran
	Outcomes	It was concluded that a significant proportion of the subjects with stroke attending the rehabilitation clinics showed depression that adversely affected their QOL
28.	Objective	This study aimed to investigate the effect of PGE2 on cholesterol oxidation product-induced neuronal cell injury. The effect of PGE2 on the toxicity of 7-ketocholesterol (7-KCS) was assessed in PC-12 cells that were differentiated following treatment with nerve growth factor. PC-12 cells exposed to 7-KCS revealed nuclear damage, decrease in the mitochondrial transmembrane potential, cytosolic accumulation of cytochrome *c*, activation of caspase-3, increase in the formation of ROS and depletion of glutathione (GSH). The mitochondria-mediated apoptotic process was evaluated by examining the inhibitory effect of PGE2 on 7-KCS-induced toxicity
	Outcomes	The results showed that PGE2 inhibits 7-KCS-induced toxicity in differentiated PC-12 cells by suppressing the mitochondria-mediated apoptotic process. PGE2 may protect against cholesterol oxidation product-induced neuronal cell injury
29.	Objective	The purpose of this study was to investigate the effect of mental practice on balance among stroke survivors. This study was a randomized experimental study in which a total of 30 subjects (16 males and 14 females) participated from the Hazrat-e-Rasul and Rofaideh Hospitals
	Outcomes	Results showed that mental practice had a significant effect on the balance among subjects with stroke in the experimental group, and it was concluded that mental practice improved balance; therefore, this can be used as a rehabilitative technique for stroke patients
30.	Objective	This study aimed to explore the role of vitamin B12 deficiency in lacunar stroke patients. In 40 first-ever lacunar stroke patients, all of whom had a brain MRI, vitamin B12 levels were determined and self-report questionnaires for fatigue and depression were completed at 3 months after stroke. Vitamin B12 deficiency was defined as a concentration < 150 pmol/L
	Outcomes	It was concluded that there was a relationship between vitamin B12 deficiency and increased levels of fatigue and depression in lacunar stroke patients. Cross-validation in a larger sample and in patients with other vascular diseases such as myocardial or cortical infarction is warranted. Positive results will justify an intervention study with vitamin B12 supplementation for those patients reporting fatigue and depressive symptoms
31.	Objective	This study aimed to evaluate the etiopathogenic role of non-alcoholic fatty liver disease (NAFLD) in vascular dementia and mixed dementia via cardiovascular pathology it may induce. The study enrolled 94 NAFLD patients referred to our clinic in Bucharest, 32 (34.05%) males and 62 (65.95%) females, 34 (36.17%) of whom were obese. Patients' cognitive status was evaluated with routine psychometric tests and their co-morbidities were also assessed
	Outcomes	It was concluded that the contribution of NAFLD in the etiopathogenic scenario of vascular dementia and mixed dementia was via cardiovascular co-morbidities. The fact entails hepatic assessment as a component of vascular dementia and mixed dementia prevention and early diagnosis

Continued

Table I. *(Cont.) Overview of experimental studies.*

Ref.		
32.	Objective	This study aimed to investigate if single nucleotide polymorphisms (SNPs) on chromosome 12p13 and within 11 kb of the *NINJ2* gene would be associated with earlier-onset (vs. late-onset) first-ever ischemic stroke and increase silent cerebrovascular lesions prior to the manifestation of stroke
	Outcomes	It was concluded that the Rs11833579 *NINJ2* SNP may bring forward the onset of first-ever ischemic stroke without increasing silent cerebrovascular lesions prior to the stroke
33.	Objective	This study aimed to investigate the cognitive function in patients with essential hypertension with different levels of vasomotor reactivity and velocity of transmembrane ion transport. One hundred and twelve patients with essential hypertension –59 women and 53 men– were examined. To estimate the vasomotor reactivity, transcranial Doppler with vasodilatory stimulus was used. Patients were screened for cognitive impairment using the Mini-Mental State Examination (MMSE), a clock-drawing test, the Frontal Assessment Battery and the Schulte test
	Outcomes	It was concluded that the development of cognitive impairment in patients with essential hypertension is associated with a decrease in cerebral perfusional reserve and a high rate of velocity of transmembrane ion transport, with the risk for cognitive impairment development in patients with essential hypertension
34.	Objective	This study reviewed the stroke database to investigate changes in carotid duplex sonographic findings, which reflect hemodynamic changes before and after carotid stenting in post-stroke dementia patients
	Outcomes	It was concluded that before, during and after carotid stenting, the flow of adjacent carotid arteries was changed in about one-third of patients, with no change in another two-thirds. The hemodynamic changes of carotid flow can be dynamic after carotid stenting and their clinical significance needs to be further investigated
35.	Objective	This study aimed to investigate the cerebral angioarchitectonic features in preclinical and clinical stages of AD. The research included 92 patients, of whom: Test group: 47 patients aged 34-79 years suffering from different stages of AD, including: –4 with preclinical stages or a high risk of disease development (CDR-0) –15 with mild dementia (CDR-1) –20 with moderate dementia (CDR-2) –8 with severe dementia (CDR-3) Control Group: 45 patients aged 28-78 years with various types of brain lesions accompanied by dementia but without AD, including: –15 with chronic cerebrovascular insufficiency of atherosclerotic genesis and moderate dementia –12 with severe vascular dementia –14 with atherosclerotic parkinsonism –4 with Parkinson´s disease
	Outcomes	Results showed that different stages of AD, changes in angioarchitectonics and microcirculation show themselves in: –lack of expressed atherosclerotic lesions of intracranial vessels in 47 (100%) patients –reduction of capillary bed in the temporal and frontoparietal regions in 47 (100%) –development of multiple arteriovenous shunts in the same areas in 47 (100%) –corresponding early venous shunts in 47 (100%) –abnormal development of venous trunks receiving blood from arteriovenous shunts in 42 (89.4%) –venous congestion at the frontal and parietal region border in 43 (91.5%) –increased loop formation of intracranial arteries in 37 (78.7%). Control group patients had no combination of such changes. It was concluded that these vascular changes are discirculatory angiopathy of Alzheimer's type (DAAT)
36.	Objective	This study aimed to develop neuropsychological screening tools designed specifically for VCI. The Brief Memory and Executive Test Battery (BMET) has been validated in relation to the ability to differentiate patients with cerebral small vessel disease from AD
	Outcomes	It was concluded that BMET is a promising screening battery for the detection of VCI and differentiation from Alzheimer's-type impairment
37.	Objective	This study aimed to establish any linkage between the MMSE score of stroke patients and clinical state
	Outcomes	It was concluded that the lower scores of MMSE in the patients were related to stroke form (hemorrhagic), aging and epileptic seizures, while the gender, parkinsonism and the time from the stroke onset appeared not to be significantly important
38.	Objective	The present study aimed to investigate donepezil (Aricept®) in dementia of diabetic origin; the efficacy is being assessed against a control group treated with placebo. The study group included 36 known cases of diabetes screened for dementia with MMSE scoring
	Outcomes	It was concluded that there is significant improvement in patients with dementia of diabetic origin following administration of donepezil hydrochloride over a period of 6 weeks

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Table I. *(Cont.) Overview of experimental studies.*

Ref.		
39.	Objective	This study aimed to investigate the incidence of dementia in a diabetic population who had no provocative or co-etiological factors other than diabetes as a causative factor for dementia
	Outcomes	Study results showed a significant positive correlation between the manifestation of dementia in diabetes and HbA1C levels in diabetes. These findings emphasize the need for absolute control over continuous near normal blood sugar levels to prevent dementia
40.	Objective	The objective of this study was to examine the clinical features of pontine infarction in the acute stage and factors affecting cognitive dysfunction
	Outcomes	It was concluded that cognitive impairment in patients with pontine infarction was not rare and may be associated with a subsequent disability handicap
41.	Objective	This study aimed to investigate the effect of solifenacin (an anticholinergic) on the cognitive function after stroke
	Outcomes	It was concluded that solifenacin did not affect the cognitive function in post-stroke patients. This might be useful information when prescribing anticholinergics to stroke patients
42.	Objective	This study aimed to explore the effect of virgin olive oil on brain lipidomics in a rat stroke model. Experimental mature male rats were treated with 0.25, 0.5 and 0.75 mL/kg/day of virgin olive oil for 30 days via gastric gavage, while the control group was treated with saline (n = 6). At the end of day 30, rat brain was removed and stored in –80 °C and lipid isolation, purification and quantitation. All lipids were analyzed qualitatively by high-performance thin-layer chromatography (HPTLC)
	Outcomes	Results showed that virgin olive oil can change brain lipidomics compared to the control group due to the presence of oleic acid and antioxidants
43.	Objective	This paper aimed to present a recent update on the role of memantine in dementia. Clinical trial registries and the database of the German Institute of Medical Documentation and Information (DIMDI) were searched to identify randomized, placebo-controlled, double-blind clinical trials with memantine, accepting original publications and meta-analyses
	Outcomes	Results of clinical trial registries, as well as meta-analyses and responder analyses, proved the beneficial effects of memantine in the treatment of moderate to severe AD
44.	Objective	This study aimed to understand the relationship between blood pressure and dementia in middle-aged women who were followed for 40 years with comprehensive medical and neuropsychiatric examinations. Among those not treated with antihypertensives, higher midlife systolic blood pressure, but not blood pressure trajectories from 1968 to 1992, was associated with dementia and AD. Those with a history of antihypertensive treatment had higher baseline systolic blood pressure than those who were never treated. In this group, those who developed dementia and AD had lower baseline systolic blood pressure and a steeper increase in systolic blood pressure from mid- to late life than those who did not
	Outcomes	Study results showed that a steeper decline in systolic blood pressure after 1992 was observed in those who developed dementia regardless of antihypertensive treatment. The association between blood pressure and dementia is complex, and influenced by antihypertensive treatment. The findings emphasized the importance for detecting increased blood pressure in midlife and to control blood pressure in those treated. Whether the trajectory of blood pressure is a risk factor or part of the clinical course for dementia needs to be elucidated. Furthermore, hypertension is a risk factor for stroke and white matter lesions, which may also lead to dementia and cognitive dysfunction. Six placebo-controlled trials on hypertension with dementia or cognitive function as outcomes have been published
45.	Objective	This was a population-based longitudinal study to show that diabetes is a risk factor for dementia, both AD and vascular dementia. The Vantaa 85+ study included 553 residents living in the city of Vantaa, Finland, aged 85 years or more, in 1991. Survivors were re-examined in 1994, 1996, 1999 and 2001. Autopsies were performed in 291 persons who died during the follow-up (48% of total population). A total of 132 of the formalin-fixed brains were also subjected to post-mortem MRI
	Outcomes	Preliminary results indicated that people with diabetes tended to develop more extensive vascular pathology, which alone or together with AD-type pathology (particularly in *APOE4* carriers) resulted in increased dementia risk. In the oldest age groups, less AD-type pathology may be needed to produce a clinical syndrome of dementia in patients with diabetes
46.	Objective	This study aimed to determine the protective role of kynurenic acid (KYNAC) on quinolinic acid-induced neurotoxicity
	Outcomes	In this study it was found that the neuroprotective potential of a new KYNAC analogue [2-(2-*N*,*N*-dimethylaminoethylamine-1-carbonyl)-1*H*-quinolinone] was represented by a significantly diminished hippocampal CA1 cell loss and preserved long-term potentiation expression. In another experiment, KYNAC played a protective role by downregulating Bax expression and maintaining mitochondrial function in MPP+-induced neuronal cell death, and it was suggested that KYNAC may have therapeutic potential in Parkinson´s disease
47.	Objective	This study aimed to demonstrate the role of oxidative stress and inflammation in APP/PS-1 transgenic mice
	Outcomes	It was concluded that adenosine deaminase presents proinflammatory and pro-oxidant proteins, with a significant decrease in antiinflammatory and antioxidant proteins in neurons and astrocytes in culture. Also in transgenic mice (APP/PS-1), a pro-oxidant and proinflammatory effect was detected compared to wild-type animals

Continued

Table I. *(Cont.) Overview of experimental studies.*

Ref.		
48.	Objective	The aim of this study was to investigate the protective effects of Mildronate® administration in rats following transient middle cerebral artery occlusion (MCAO). Male Wistar rats were subjected to MCAO for 90 minutes, followed by intraperitoneal administration of Mildronate® at doses of 100 and 200 mg/kg for 14 days. The beam-walking, rotarod and cylinder tests were used to assess sensorimotor function, vibrissae-evoked forelimb-placing and limb-placing tests examined responses to tactile and proprioceptive stimulation. Following behavioral testing, the brain tissue infarct volume was measured
	Outcomes	The results showed that saline-treated MCAO rats had minor or no spontaneous recovery in sensorimotor and proprioceptive function up to 14 days post-stroke. Treatment with Mildronate® at a dose of 200 mg/kg was found to accelerate recovery of motor and proprioceptive deficits in limb-placing, cylinder and beam-walking tests. Infarct size did not differ among the experimental groups on post-stroke day 14. It was suggested that Mildronate® treatment improves the functional outcome in MCAO rats without influencing the infarct size
49.	Objective	This study aimed to explore the relationship between obesity and cognitive function
	Outcomes	It was concluded that a high body mass index and waist circumference lead to decrease of cognitive function. It is important to assess cognitive function by using more sensitive methods in such patients
50.	Objective	This study aimed to explain that the vascular component plays a significant role in VCI and is likely to influence the clinical phenotype of dementing diseases
	Outcomes	It was suggested that white matter abnormalities together with microstructural tissue abnormalities play an important role. Definition of the crucial lesion types is the prerequisite for development and validation of VCI criteria. This will ultimately create the basis for intervention trials in VCI or subtypes of VCI according to lesion pattern
51.	Objective	This study aimed to explore the clinical–psychological characteristics of chronic cerebral ischemia (CCI) patients. Two hundred and nine CCI I-III patients (aged 52 ± 10.7 years), 93 with constitutional phlebopathy and 116 without it
	Outcomes	It was concluded that in CCI patients, different disease manifestations are due to constitution; therefore, in stage I the harmonious type of attitude to illness appeared more often. For patients to "get accustomed" to disease manifestations and cease to notice them, CCI progression did not change the attitude to illness and treatment adherence. As disease manifestations are new to patients, they analyze and endure a semiology increase that in later CCI stages leads to realization of disease severity, the desire for active contribution to treatment success, an increase in treatment adherence and the number of patients with a harmonious type of attitude to illness
52.	Objective	This study aimed to examine: 1) the association between cerebral small vessel ischemic disease and retinal microvessel behavior; 2) the relationship between retinal blood vessel reactivity and measures of cerebrovascular function. In a cohort of 12 patients with ischemic white matter disease and 10 healthy controls (aged 38-85 years), retinal vasoreactivity was measured following high-frequency flicker light stimulation. Middle cerebral artery vasoreactivity was measured using transcranial Doppler ultrasound and 1.5 Tesla brain MRIs were reviewed
	Outcomes	Results showed that patients with cerebral small vessel disease have significantly attenuated retinal venous and arterial vasoreactivity, and it was concluded that impairment of retinal microvascular function is associated with ischemic white matter disease and measures of cerebral vascular function. Microvascular dysfunction in the eye may predict cerebral small vessel disease
53.	Objective	This study investigated the cortical thickness changes over 3 months in a group of stroke patients compared with controls. Patients with acute hemispheric stroke were studied within 2 hours and serially over 3 months and the acute and 3-month scans were compared with independently acquired control images. High-resolution isotropic MPRAGE images were analyzed using Freesurfer V5, comparing global average cortical thickness, hippocampal and thalamic volumes. Individual participant change scores between timepoints were computed and converted to percent change scores. Twelve stroke patients (9 men; 7 left-hemispheric; mean age 65.1 years) and 10 control participants (5 men; mean age 67.2 years) were included
	Outcomes	This study concluded that post-stroke changes in regional cortical thickness are demonstrable even over short timeframes. Contralesional cortical thickness increases may represent compensatory mechanisms. Significant reductions in thalamic volume may represent evidence of early post-stroke atrophy
54.	Objective	The objective of the study was to examine the prevalence of post-stroke depression (PSD), its relation to cognitive impairment and functional outcome. The data from 200 patients with acute stroke and 6, 12 months and 2 years after stroke have been evaluated. Patients were tested with the Hamilton Depression Scale (HDS), MMSE, Barthel index and Rankin scale. All patients underwent neuroimaging and MRI
	Outcomes	Results showed that depressive states were observed in 17% in the acute stage of stroke, in 35% after 6 months, 43% at 1 year and 56% at 2 years after stroke. In 48% of patients with ischemic stroke the focus was located in the right hemisphere, and in 52% in the left hemisphere. Cognitive impairment, neurological and physical complications, as well as everyday life dependence, were more pronounced in patients with depression than those without it, and this progressed over time. It was concluded that a significant share of patients have PSD and its frequency increases by the 2nd year of life. In the post-stroke period, all patients need psychological adjustment due to a direct correlation between the development of depression and the outcome of stroke (cognitive and neurological disorders)

Continued

Table I. *(Cont.) Overview of experimental studies.*

Ref.		
55.	Objective	This study was designed to investigate the relationship between diabetes and dementia in elderly primary healthcare patients. The study included 409 diabetic patients over 60 years of age, registered with 3 family medicine practices, in the region of Sarajevo and Banja Luka, BiH. As a part of the annual screening for chronic complications of diabetes, the screening for dementia was conducted using MMSE, with the scores adjusted for age and education. Questionnaires were evaluated, and the patients with a score of < 26 were invited back to the clinics for additional evaluation
	Outcomes	Results: 33% of diabetic patients reached the criteria for vascular dementia and 44% for mild cognitive impairment. Length of the disease and gender were not significantly correlated with the score on MMSE. It was concluded that diabetes increases the incidence of mild cognitive impairment and vascular dementia

America. Costs were attributed to informal (unpaid) care, direct costs of social care (provided by institutions and community care professionals) and the direct costs of medical care (treatment in primary and secondary care). Costs of informal care and the direct costs of social care generally contribute similar proportions of total costs, while the direct medical costs are much lower (1).

BIOMARKERS IN VASCULAR COGNITIVE IMPAIRMENT

The pathogenesis of central nervous system (CNS) disorders such as vascular dementia, stroke, Alzheimer's disease, Parkinson's disease or acquired brain injury is very complex and involves several molecular mechanisms. Since there is no disease-modifying treatment to date for these disorders, the development of multimodal drugs able to act on different targets might provide some advancement in this regard. Molecules contributing to neuronal apoptosis and degeneration, a common finding in all these medical conditions, constitute drug targets of particular interest. Excitotoxicity, inflammation, oxidative damage, apoptosis, neurodegeneration and neurotrophic alterations have a relevant pathogenetic role in all these CNS disorders. Glutamate, calpain, proinflammatory cytokines such as TNF-α, reactive oxygen species, amyloid and tau proteins, neurotrophic factors like brain-derived neurotrophic factor (BDNF), insulin-like growth factor I (IGF-I) or vascular endothelial growth factor (VEGF), and signaling proteins including kinases such as protein kinase Akt, glycogen synthase kinase-3 beta (GSK-3 beta) and cyclin-dependent kinase 5 (CDK5) are some of the key molecular players in the pathophysiology of heterogeneous CNS disorders. Therefore, drugs modulating multiple mechanisms mediated by these molecules appear to be a promising treatment option for such disorders. The multimodal effects of neurotrophic factors, erythropoietin, statins or Cerebrolysin, a peptidergic compound mimicking the activity of endogenous neurotrophic factors, were investigated in different experimental models of stroke, Alzheimer's disease and traumatic brain injury with positive results. Clinical studies also demonstrated the efficacy of such an intervention in patients with vascular dementia (2).

METABOLIC SYNDROME AND VASCULAR DEMENTIA

The metabolic syndrome is a cluster of risk factors predisposing to vascular disease and diabetes, but also to cognitive impairment and dementia. It is unclear, however, if the metabolic syndrome is associated with a specific clinical profile or different prognosis among patients with cognitive complaints or early dementia (3).

Population-based longitudinal studies have shown that diabetes is a risk factor for dementia, both Alzheimer's disease and vascular dementia. As diabetes increases cerebrovascular disease, its association with vascular dementia is understandable. Some mechanisms have been suggested for the link with Alzheimer's disease, such as vascular mechanisms, toxic effects of hyperglycemia, insulin resistance of the brain or the formation of advanced glycation end products. Cerebrovascular and neurodegenerative pathologies often occur together and influence each other, especially at older ages. Studies on diabetes and Alzheimer's disease have thus had conflicting results: no association between diabetes and Alzheimer's disease pathology, or less tau and amyloid pathology in patients with diabetes, or more pronounced Alzheimer's disease-type changes in the hippocampus of *APOE4* carriers with diabetes (4).

POST-STROKE COGNITIVE IMPAIRMENT

Stroke is a stressful, life-threatening experience, being intensified by its sudden and unanticipated nature, affecting the patient's sense of wholeness and safety, and leaving him or her with a lasting sense of vulnerability. At the psychological level, any life-threatening experience accompanied by an emotional response, including intense fear, helplessness or horror, may lead to various post-traumatic stress disorder (PTSD)-like symptoms. The cognitive consequences of PTSD include impairment of learning and recall, verbal and working memory, attention and self-referential semantic processing. The hypothalamic–pituitary–adrenal (HPA) axis is largely responsible for controlling stress reactions. Cortisol is an output of the HPA axis with binding affinity for cerebral glucocorticoid receptors. Limbic regions, such as the hippocampus, amygdala and prefrontal cortex, are targeted by glucocorticoids and are essential for producing adapted and integrated neuroendocrine and behavioral responses to stress. Acute stress is associated with inhibition of hippocampal neurogenesis and loss of neuronal branching in the hippocampus. Finally, the acute phase of stroke is associated with increases in the release of acetylcholine (ACh) and circulating proinflammatory cytokines, with recent studies demonstrating causal links between inflammatory pathways and cholinergic signaling. The cholinergic antiinflammatory pathway may be mitigated by acetylcholinesterase (AChE) and the closely related enzyme butyrylcholinesterase (BChE), both of which hydrolyze and inactivate ACh. It was shown that serum AChE and cholinergic status measurements can predict neurological outcome, survival and inflammatory reactions following acute ischemic stroke and an inverse correlation

between bedtime saliva cortisol levels, hippocampal volume and cognitive scores measured 6 and 12 months post-stroke. Patients with larger hippocampi had significantly better cognitive results 6 and 12 months post-stroke, suggesting that stroke patients with smaller hippocampi may be prone to develop cognitive decline. We hypothesize that small hippocampal size might be the result of prolonged exposure to neurotoxic levels of cortisol secreted during chronic stress. The stress-related (PTSD-like) mechanisms could have particularly profound long-term consequences when they meet an already damaged brain after an ischemic stroke. Therefore, stress, as well as secondary hippocampal/limbic lesions, may be devastating contributors to a poor post-stroke prognosis. The "stressogenic-vulnerable patient" is prone to develop cognitive impairment. Hence, detection of a maladaptive stress response in stroke patients has the potential to provide new concepts for targeted intervention aimed at slowing post-stroke cognitive and affective deterioration (5).

VASCULAR DEPRESSION

The prevalence of cerebrovascular disease and depression increases with age. Therefore, the frequency of their combination is quite high and inevitably the question arises: is this comorbidity accidental or does it have certain pathogenetic mechanisms? Until the mid-20th century, the fact that cerebrovascular disease can lead to depression has received some clinical evidence, based on the identification of depression in patients with cerebrovascular disease and attempts to identify specific features of such states. However, these concepts could not be confirmed by findings of the pathogenetic link between the mentioned pathological processes, and the term "vascular depression" disappeared from the psychiatric literature. The creation of new notions of "vascular depression" is based on a high incidence of various ischemic symptoms, including "silent" ischemic attacks, in neuroimaging studies of patients with depression. It was established that, in cases of depression, stroke is 6.4 times more likely to occur than in the average population, and stroke, in turn, increases the risk of depression 2.3 times. The symptoms of "vascular depression" are characterized by certain peculiarities. In general, the combination of late-life depression and cerebrovascular disease is a complex problem and its solution must involve both psychiatrists and neurologists, which largely predetermines the effectiveness of the treatment of such patients (6).

NEUROREGENERATION IN NEURODEGENERATIVE DISORDERS

Neuroregeneration is a relatively recent concept that includes neurogenesis, neuroplasticity and neurorestauration–implantation of viable cells as a therapeutic approach. Neurogenesis and neuroplasticity are impaired in the brains of patients suffering from Alzheimer's disease or Parkinson's disease, and correlate with low endogenous protection as a result of diminished growth factor expression. Therefore, it was hypothesized that the brain possesses, at least in the early and interim stages of the disease, a "neuroregenerative reserve" that could be exploited by growth factors or stem cell/neurorestauration therapies, which may open new avenues for the treatment of Alzheimer's disease and other neurodegenerative disorders (7).

CONCLUSIONS

Vascular dementia comprises a range of cognitive disorders related to cerebrovascular disease and its understanding has evolved substantially in recent years based on preclinical, neuropathological, neuroimaging, physiological and epidemiological studies. The emergence of sophisticated genetic and molecular tools, combined with imaging techniques of unprecedented spatial and temporal resolution, and their application to in vivo models, provides an appropriate approach to further our understanding of this entity and to better characterize its neuropsychological profile, as well as to engage interested clinicians and researchers in providing better care for patients whose vascular disease affects cognitive function.

DISCLOSURES

The authors state no conflicts of interest.

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