Mini Review

Vascular dementia: current considerations

Abstract

Vascular dementia (VaD) is the second neuropathology that causes dementia in older adults, a reason for this classification, diagnosis and management of the child of great importance worldwide. It is classified into: dementia of multiple infarctions, post-stroke dementia, subcortical ischemic vascular pathology, dementia due to small vessel disease, dementia of hypoperfusion and hereditary vascular dementia. Contrary to what is thought, today this pathology is a problem due to partial ignorance of its pathogenesis, where Alzheimer's disease could be participating, thus constituting a mixed dementia. Combined, there is no treatment that stops its progressive progress, however, it is used as: donezepilo, rivastigmine and galantamine, those that have cognitive functions. New research related to rescue and ornithine, despite this, the curative treatment of the disease remains uncertain.

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Introduction

The third part of elders at 85 years of age will have some type of dementia, all affected, 16% can be classified as vascular dementia (VaD), for this reason, it is considered the second neuropathology causing dementia in older adults.[1] It defines a gross mode with the presentation of the problems of reasoning and the commitment of the planning, judgment and memory, product of an alteration in the secondary cerebral circulation a cerebrovascular accident.[2] Estimates a prevalence in Latin America of 0.6% To 2.1% For individuals > 65 years1; In the United States, a prevalence of 0.98%, between 80-89 years of 4.09% and in> 90 years of 6.10% is estimated in 71-79 years, demonstrating that cerebrovascular dysfunction progressively increases after the age of 85 Years It is believed an association between Alzheimer's disease and vascular dementia can be considered, considering a mixed dementia.[3]

The risk factors for vascular dementia are chronic hypertension,4 diabetes mellitus, smoking, APOE[4] isoforms, obesity, age[5] late-onset depression[6] and hypercholesterolemia.[7] The onset of VaD is due to any vascular injury (ischemia) That affects brain functional tissue causing gliosis and demyelination. Atherosclerosis, thrombosis or valvulopathies are the direct causes of ischemia,[8] depending on the origin of the lesion we include a spectrum of VaD subtypes such as multi-infarct dementia, post-stroke dementia, dementia due to disease of small vessels subcortical ischemic vascular disease, dementia Of hypoperfusion and hereditary vascular dementia.[9] The dementia due to disease of small vessels is subclassified in Binswanger's disease, amyloid angiopathy with hemorrhages, CADASIL (Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarts and Leukoencephalopathy).[10]

Therefore, it is estimated that the causal origin of cognitive deterioration are cerebrovascular disorders; The cerebral blood vessels are fundamental for the supply of oxygen and nutrients, in addition, they participate in the trophic signaling that establishes connections between the neurons and the glia with the cerebrovascular cells, therefore, the vascular damage interrupts homeostatic interactions, focusing on the white matter, area of greatest risk of vascular damage.[11] The morphological changes characteristic of VaD involve globally: infarcts in cortical and subcortical arterial territories, lesions in the aforementioned white matter, and the hippocampus[12] this is translated into progressive deterioration of memory, executive dysregulation (difficulty in planning and organizing), aphasia (Dysfunction in the ability to produce and understand complex movements and gesticulations), agnosia (difficulty in visuospatial processing), neuro-conductive symptoms (agitation, apathy and disinhibition)[13] disability, Depression and behavioral changes.[14]

For the diagnosis of Vascular dementia, the NINDS - AIREN criteria described in Table 1[15,16] are used. New studies support the use of biomarkers as a diagnostic guideline, among which microRNAs have been proposed: miR-409-3p , MiR-502-3p, miR-486-5p and miR-451a, found in plasma of patients with VaD and cerebrovascular disease of small vessels[17] however, Quinn, et al. show that there is no strong evidence for the early use of biomarkers in the diagnosis of VaD, equally maximizing the importance of the standard diagnostic approach and strategy, which has worked well.[18]

Table I Diagnostic criteria of vascular dementia taken from NINDS – AIREN

Possible vascular dementia	Vascular dementia likely	Vascular dementia defined
Focal, non-neuroimaging neurological signs that exhibit cerebral vascular disease.	Prematurity of gait.	Criteria for probable vascular dementia.
No temporal association between dementia and stroke.	Reports of instability and frequent falls.	Vascular disease confirmed by biopsy or necropsy.
Dementia onset with evidence of cerebral vascular disease.	Urinary urgency.	No histological lesions suggestive of Alzheimer's disease (although there may be mixed dementia).
	Psuedobulbar palsy.	Absence of other diseases as a cause of dementia.
	Personality and mood with evidence of alterations.	

With respect to the treatment, the acetylcholinesterase inhibitors are used with unusual expectations, such as: Donezepilo, Rivastigmine and Galantamina, also N-methyl-D-Aspartate inhibitors like memantine are used, however, their effectiveness Has not been shown to be sufficient to eliminate VaD -derived disorders.[19] Controversially, some authors affirm that the aforementioned drugs lack treatable and identifiable therapeutic objectives, this being sustainable in the real ignorance about the injection of cerebrovascular deterioration with the secondary cognitive disorder.[20]

Studies have therefore been carried out to identify new molecules involved in the development of the disease. One of these studies describes the use of nanoparticles of Solid lipids loaded with Resveratrol (R-SLNs) in murine for reduction of mitochondrial reactive oxygen, lipid peroxidation and protein carbonyls. In addition, there is a decrease in levels of hypoxia-inducible factor 1α (HIF-1α). In this way, R-SLNs promises to be an innovative therapeutic strategy in VaD.[21] Other investigations suggest the use of psychostimulants such as citicoline and vinpocetine, supplements as actovegin, and finally, therapies with herbal derivatives, within these the huperzine A, however, its favorable effects are not clearly elucidated. Lifestyle modifying therapies, a balanced diet, exercise and control over vascular risk factors, it should not be forgotten that their execution plays an important role in the prevention and treatment of VaD.[22] Finally to understand that vascular dementia is a pathology of difficult management and uncertain prognosis, to date only treatable to improve the cognitive abilities until the causal factor of the disease degrades them volitionally.

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Conflict of interest

Author declare that there is no conflict of interest in the doing of the present manuscript.

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