COGNITION AND VASCULAR HEALTH

COGNITIVE DECLINE AND VASCULAR DEMENTIA

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Abstract

It is widely recognized that strokes are the primary cause of acquired disability, the third leading cause of death, and a major contributor to dementia. Vascular dementias, collectively referred to as post-stroke dementia, encompass various conditions that develop after a stroke, including:

Multi-infarct dementia (MID)

Vascular dementia (VaD)

Vascular cognitive impairment (VCI)

Post-stroke cognitive impairment

This article aims to review the different forms of vascular dementia, their risk factors, and the diagnostic criteria associated with these conditions.

***Key words:** Stroke, vascular dementia, Risk factors Diagnosis*

Introduction

Post-stroke dementia is a major cause of disability and dependency.The prevalence in patients surviving after a stroke is 30% and the incidence tends to increase after the vascular event from 70% in the first year to 48% 25 years later. (**1)**

Vascular dementias represent the second cause of dementia after Alzheimer's disease (AD).

In recent years, the concept of "cognitive disorders of vascular origin" has been preferred , which includes vascular dementias proper and mild non-dementia cognitive disorders of vascular origin.

Their diagnosis nevertheless remains difficult due to the diversity of vascular lesions responsible for cognitive disorders and their frequent association with AD.

The neuropsychological profile is of the subcortico-frontal type with more marked impairment of executive functions, with the possibility of impairment of more cortical functions depending on the location of the vascular lesions.

In addition to vascular injury, risk factors for the occurrence of post-stroke dementia have been described:

1. Vascular risk factors (hypertension, hypercholesterolemia, diabetes, atrial fibrillation, smoking, heart failure, obesity and physical inactivity).
2. Advanced age at the time of onset of disorders.
3. The socioeconomic level.
4. Rankin score before stroke.

Definitions

1) Cognitive decline: The term cognitive decline without dementia defines the acquired loss of cognitive functions without impact on daily life.
2) Dementia syndrome: the term dementia syndrome designates an authentic memory disorder associated with at least one other disorder of higher functions (language, praxis, gnosia, executive functions).

The diagnosis of dementia is established according to the criteria of DSMIIIR **(2)** and ICD10*. **(3)**

Epidemiology

Prevalence: second leading cause of dementia after Alzheimer's disease in Western countries **(4.5)** and probably the leading cause of dementia in developing countries.

Epidemiological data are very heterogeneous due to diagnostic difficulties, in the absence of clearly established anatomoclinical criteria, both clinical and neuropathological, on the one hand, and on the other hand by their association with Alzheimer's disease.

The incidence is estimated at 2.5/1000 inhabitants. As for the prevalence, it increases with age.

Overall, the prevalence of dementia after stroke is 3.5 to 5.8 times higher than in the non-stroke population. (**6,7)**

This diagnostic difficulty has led specialists to describe post-stroke dementias including Alzheimer's dementia, the preclinical phase of which is shortened by the stroke, and vascular dementias. **(8)**

Risk factors

The risk factors are those of strokes

1) HBP: major and established risk factor for stroke where the risk is multiplied by 4.

   HBP also contributes to the risk of developing cognitive impairment and dementia. (**9,10)**

   An increase in diastolic and/or systolic blood pressure, especially if left untreated, would be at high risk of developing white matter lesions and therefore cognitive disorders and dementia. **(11; 12)**

2) Diabetes: Diabetes through macro and microvascular lesions and changes in carbohydrate and lipid metabolism increases the risk of multiple ischemic lesions and the onset of cognitive disorders and dementia.

Vascular. (**13)**

3) Other risk factors

Hypercholesterolemia

Heavy alcohol consumption **(14)**

Hyperhomocysteinemia **(15,16)**

Thromboembolic heart diseases (atrial fibrillation, valvulopathies, heart failure)

Coagulation disorders **(17,18)**

Diagnosis

As previously written, the diagnosis of vascular dementia is difficult in the absence of defined neuropathological criteria .

It is based on the following arguments:

- Clinics (neurological and neuropsychological examinations)
- Radiological: brain imaging data

But the diagnosis of certainty is anatomopathological

- ***Clinical arguments***

1) Clinical diagnosis: Onset of cognitive disorders when walking up stairs

A fluctuating evolution

Gait and balance problems

At a more advanced stage:

Akineto- rigid parkinsonian syndrome

A pyramidal attack

A pseudobulbar syndrome

Sphincter disorders

2) Neuropsychological examination

A dementia syndrome under frontal corticosteroid with impairment of executive functions

Difficulty recalling memories

Episodic memory disorders affecting information retrieval processes with effective cueing and preserved recognition

Verbal fluency is poor

Language, praxis and gnosia depend on the presence of cortical lesions such as lobar hemorrhages and cortical infarcts.

- ***Radiological arguments***

Imaging

The diagnosis of vascular dementia can only be made with the help of quality imaging. Indeed, normal imaging can eliminate the diagnosis.

The examination of choice is brain MRI with all these sequences:

- FLAIR and T2 for ischemic lesions including small lacunae and signs of leukoaraiosis that may be missed on CT. (figure 1)
- The T2* sequence for the visualization of old or recent microhemorrhages. (figure 2)

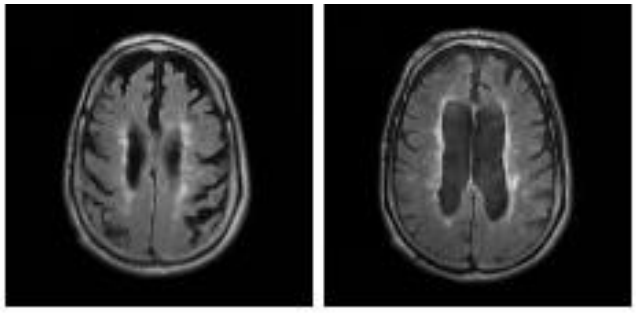


Figure1 :Brain MRI FLAIR sequence

Diffuse hypersignals of the periventricular and subcortical white

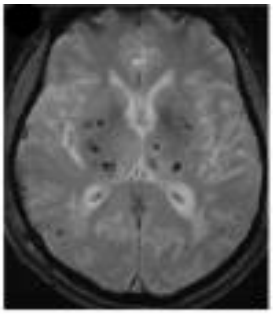


Figure 2 : Brain MRI T2 sequence Hypertensive microangiopathy

Diagnostic criteria

The evolution of the criteria for vascular dementia from the 1960s to the present day has seen the proposal by specialists of different diagnostic tools such as the Hachinski score * in 1975 **(19)(** table 1), ADDTC **(20),** ICD10* (table 2) and NINDS AIREN* **(21)** (table 3) and DSMIV* **(22)** (table 4).

Each tool has its advantages and disadvantages, especially in terms of sensitivity, hence the diagnostic difficulty. Although for many authors the Hachinski scores and the NINDS AIREN have the best sensitivity and specificity respectively **(23).**

In practice, to distinguish multiple infarction dementia from Alzheimer's dementia according to the Hachinski score: the presence of hypertension, a history of stroke, focal signs and a fluctuating stair-step progression are highly suggestive.

Clinical forms

1) Multiple infarct dementia (figure 3)

Multiple infarct dementias are characterized by the presence of multiple cortical and subcortical infarcts of cardiovascular cause.

The clinical picture combines cognitive deficits of the aphasic, agnosic, apraxic and/or dysexecutive type associated with motor or sensory signs.

2) Dementia due to strategic infarction

In this case, dementia is linked to the location of the infarction.

The most incriminated locations are:

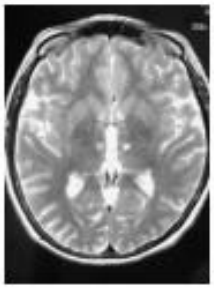
Bithalamic infarction (figure 4) combines severe memory disorders, apathy, emotional indifference and attention disorders.

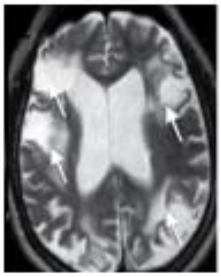
Bitemporal infarction

Bifrontal infarction

Infarction due to involvement of the dominant hemisphere (figure 5)

Brain MRI T2 sequence





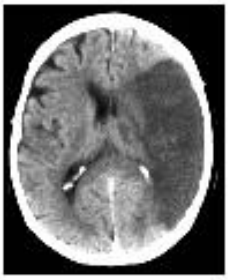


Figure 3: Bithalamic Infarction **Figure 4:** Multiple Stroke **Figure 5:** Left hemisphere Stroke

In addition to these primary localizations, cognitive disorders have also been reported in small infarcts located in functional areas such as the caudate nucleus, the anterior arm of the internal capsule, the medial temporal lobe and the cingulate gyrus.

3) Vascular dementia due to cerebral microangiopathies

Cerebral microangiopathies responsible for lacunar infarctions or intracerebral hematomas are currently one of the main causes of vascular dementia.

They include:

a) Ischemic subcortical dementias associate gaps and focal or diffuse lesions of the periventricular white matter (figure 1) this is the classic radiological description of Hachinski : leukoaraiosis

These white matter lesions may be secondary to vascular causes with risk factors such as age, hypertension and diabetes.

Or of genetic origin: CADASIL, CARASIL and MELAS

- **CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy):** it is an autosomal dominant

genetic disorder that affects the small intracerebral arteries secondary to a mutation in the NOTCH3 gene located on chromosome 19q12. (**24,25)**

Clinic: age of onset

Recurrent lacunar stroke with focal signs, pseudobulbar syndrome, frontal corticosteroid dementia syndrome and mood disorders.

In about 30% of cases, migraines are present.

- **CARASIL (cerebral autosomal recessive angiopathy with subcortical infarcts and leukoencephalopathy):** mainly described in Japan , it combines:

  Recurrent strokes and TIAs

  Ophthalmoplegia

  Facial paralysis

  Bone damage with calcification of the ligaments.

- **MELAS (mitochondrial myopathy , encephalopathy , lactic acidosis and stroke- like episodes):** cytopathy mitochondrial which associates myopathy, encephalopathy and pseudo strokes.

b) Hemorrhagic dementias

They are secondary to sclerohyalinosis which is defined by a thickening of the walls of the small terminal arteries following chronic hypertension or unbalanced diabetes; this results in hematomas or microbleeds fig 2.6). (**26)**

It is the multiplication of these lesions which is responsible for cognitive disorders.

Clinically, the dementia syndrome is of the corticofrontal type with slowness of ideas , apragmatism, a depressive syndrome and emotional lability. **(27)**

The lacunar state often described is linked to the multiplicity of lesions on the corticospinal tracts .

There may be a pseudobulbar syndrome due to damage to the 2 corticobulbar geniculate bundles .

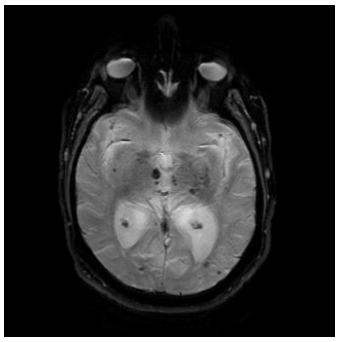


Figure 6: Brain MRI T2* sequence microbleeds

4) Mixed dementias

They correspond to the association of degenerative dementia (Alzheimer's) and vascular dementia. **(28)**

This association would be linked to the coexistence of common risk factors between Alzheimer's disease and vascular dementia: HTA, Ɛ4 allele of Apolipoprotein E , diabetes and atherosclerosis. **(29)**

For some authors, senile plaques and neurofibrillary degeneration would be the result of an ischemic process **(30,31,32).**

The diagnostic criteria are not very specific and sensitive; it is therefore very difficult to differentiate vascular dementia from mixed dementia.

The peculiarity of mixed dementias is the frequency of depressive syndrome, gait disorders and the existence of focal signs.

Evolution

The evolution remains unpredictable due to comorbidities and cardiovascular events that may occur.

Some studies **(33)** have shown that the evolution of MMSE is less rapid than in Alzheimer's disease.

Treatment

There is no proven treatment other than primary prevention. Early management of hypertension would reduce the risk of developing dementia **(11, 34,35),** without forgetting other risk factors.

Secondary prevention also has its place because even if the stroke has a strategic location, managing the risk factors and the direct cause can prevent a recurrence of stroke and therefore reduce the risk of developing dementia.

Primary prevention is essential. Early management of high blood pressure has been shown to reduce the risk of developing dementia. Other modifiable risk factors (diabetes, tobacco, alcohol, dyslipidemia, etc.) must also be taken into account, as well as the cause of strokes when possible (antiphospholipid antibodies , etc.). After a stroke, the most rigorous possible treatment of risk factors is essential, particularly that of high blood pressure. It is advisable to reduce the blood pressure of any patient who has suffered a cerebral infarction, even in normotensive patients . Thus, the PROGRESS study showed a reduction in the number of patients developing cognitive disorders when they were treated with the combination perindopril / indapamide , it also appears that white matter abnormalities often associated with vascular dementia are improved by such treatment. When dementia is established, other treatments may be proposed, but none of them have marketing authorization for this indication.

Conclusion

Vascular dementias, due to their clinical and etiological heterogeneity, are difficult to diagnose. Their frequent association with Alzheimer's disease is the main problem. While waiting for more sensitive and specific diagnostic criteria, optimal management of risk factors, especially in primary prevention, is strongly recommended.

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Table 1: Hachinski Ischemic Score (19)

Symptom	Score
Brutal Installation	2
Aggravation by steps	1
Fluctuating Evolution	2

High blood pressure	1
History of stroke	2
Signs of atherosclerosis	1
Focal neurological symptoms	2
Focal neurological signs	2
Night confusion	1
Relative preservation of personality	1
Depression	1
Somatic complaints	1
Emotional lability	1
Interpretation: Degenerative dementia if <4; vascular dementia if >7; mixed dementia if between 4 and 7.	

Table 2: Vascular Dementia According to the International Classification of Diseases (ICD-10)

Code	Description
F01	Vascular dementia due to brain infarction caused by cerebrovascular disease or hypertension. Effects are cumulative.
F01.0	Acute onset vascular dementia: Rapid onset following repeated strokes or vascular thromboses, embolisms, or hemorrhages. Rarely a single massive infarction.
F01.1	Vascular dementia due to multiple infarctions: Progressive onset after numerous transient ischemic episodes leading to cortical gaps.
F01.2	Subcortical vascular dementia: History of hypertension with ischemic destruction in deep white matter; the cortex remains mostly unaffected.
F01.3	Mixed vascular dementia: Both cortical and subcortical involvement.
F01.8	Other forms of vascular dementia.
F01.9	Vascular dementia, unspecified.

Table 3. Diagnostic Criteria for Vascular Dementia according to the National Institute of Neurological Disorders and Stroke–International Association for Research and Education in Neuroscience (NINDS-AIREN) (21)

Criterion	Description
I – Probable Vascular Dementia (all must be present)	Dementia (memory deficit + decline in two or more cognitive areas, significant impact on daily life). Exclusions: delirium, psychosis, severe aphasia, neurological deficits impeding

	assessment, Alzheimer's disease. Cerebrovascular pathology (focal signs or radiological evidence on CT/MRI). Temporal relationship suggested by sudden/fluctuating deterioration or onset within 3 months of a recognized stroke.
II – Clinical Manifestations Suggesting Probable Vascular Dementia	Early gait disturbances, balance problems, urinary disorders, pseudobulbar palsy, mood disorders (abulia, depression, emotional incontinence, dysexecutive disorder).
III – Signs Suggesting Unlikely Vascular Dementia	Early memory disorder with progressive worsening, absence of focal signs, absence of vascular lesions on brain imaging.
IV – Possible Vascular Dementia	Dementia with focal neurological signs but without vascular lesions or clear temporal relationship between dementia and stroke.
V – Certain Vascular Dementia	Probable vascular dementia + histopathological evidence (biopsy/autopsy). Absence of neurofibrillary degeneration and senile plaques beyond normal aging. Absence of other pathological conditions.

Table 4. Diagnostic Criteria for Vascular Dementia according to the Diagnostic and Statistics Manual for Mental Disorders (DSM-IV) (22)

Criterion	Description
A. Cognitive Deficits	1. Memory impairment (difficulty learning new information or remembering previously learned information)
	2. One or more of: aphasia, apraxia, agnosia, or disturbances in executive functions (planning, organizing, abstract thinking)
B. Impact on Functioning	Significant impairment in social or professional functioning, with a notable decline compared to prior functioning
C. Focal Neurological Signs	Focal neurological signs or symptoms, or cerebrovascular disease found in imaging (e.g., multiple infarcts)
D. Delirium Exclusion	Deficits do not occur exclusively during delirium