

## EVOLUTION OF THE CONCEPT OF VASCULAR DEMENTIA

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*[L'evoluzione del concetto di demenza vascolare]*

### ABSTRACT

The concept of vascular dementia has had many evolutions during the years. The importance of knowing the latest developments about terminology and concept of vascular dementia, depends from many aspects in field of prevention, diagnosis and therapy.

**Key words:** *Vascular dementia, multinfarct dementia, vascular cognitive impairment, vascular cognitive disorder.*

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The onset of a cognitive impairment with aging is a common evidence that has led, since the time of Hippocrates and Pythagoras, to study mechanisms that come into play in senility. However, only in seventeenth century prepare the ground to understand the complex world of dementia. In that time, in fact, born the idea that behind cognitive impairment of old age is a type of vascular damage, most frequently ischemic rather than hemorrhagic. Just in '600 then start talking of vascular dementia. To describe for the first time clinical vascular dementia in detail was Thomas Willis who, in 1672, described it as "dullness of mind and forgetfulness followed by stupidity and foolishness", assigning liability to two main causes: aging and vascular disease. But to get a better understanding of pathophysiology underlying dementia, we must wait until the end of nineteenth century, in which Otto Binswanger and Alois Alzheimer published two studies, independently apart, on cerebrovascular dementia: a form characterized by brain atrophy arteriosclerosis, attributable to milestones apoplexy but probably due to multiple lacunar stroke, and a

form with injuries of subcortical white matter and dilatation of ventricles which later took the name of Binswanger's disease. In 1910, Kraepelin, based on works of Alzheimer and Binswanger, proposed term "arteriosclerotic dementia" to refer to main form of senile dementia; instead dementia described by Alzheimer in 1907, which from him later took the name, was included among the presenile dementias. This approach, if allowed further insight of pathogenesis, however, represented a misunderstanding, as it appealed to the term "arteriosclerotic dementia" as a synonym for "senile dementia".

The term "arteriosclerotic dementia" meant a slow progression cognitive impairment, due to a state of chronic brain ischemia by diffuse atherosclerosis of small cerebral vessels. Just to emphasize chronicity and progression of this process, Binswanger called it "chronic progressive encephalitis", ascribing the cause to alterations of cerebral perforating vessels followed by formation of lacunae or infarcts and deep white matter demyelination without involvement of cortical gray substance; together with these last lesions are generally associated ventricles enlargement, aphasia,

hemianopsia and hemiparesis<sup>(1)</sup>. This framework, which later take its name from Binswanger, actually does not reflect different range of vascular dementia, but it is only a part. In any case, there was common accordance on the main pathogenetic moment: an arteriosclerotic narrowing of cerebral blood flow.

In the Seventies, in response to growing evidence about frequent involvement of great vessels in vascular dementia, Vladimir Hachinski advanced the hypothesis that primary mechanism not resided in a cerebral small vessels damage, as in a disease (mostly atheromatous) of great vessels, responsible for genesis of many cerebral infarctions, small or large. To describe this framework and, at the same time put the emphasis on progressiveness of phenomenon, in 1974 Hachinski coined term Multi-Infarctual Dementia (MID)<sup>(2)</sup>.

For nearly two decades, MID has been an undisputed synonym of vascular dementia. Clinical case of MID is characterized by acute onset and often “stepped” trend with worsening followed by phases of stabilization and sometimes by apparent improvement, followed by new sudden deterioration (due to new ischemic event). It is characterized by cognitive deficits (memory, strategic skills, mental flexibility) frequently accompanied by focal signs and symptoms due to extensive lesions affecting cortical areas. Focal neurological deficits are thus indicative of affected territory.

These clinical features, however, did not correspond with other forms of cognitive vascular impairment, characterized by the presence of vascular subcortical lesions (lacunae and white matter lesions). These lesions are the result of small cerebral vessels alteration and would be responsible of a clinical framework quite different from that of MID. The group of subcortical ischemic vascular dementia (SIVD), as it was named, is characterized by an insidious onset, slowly progressive, non “stepped”, course; impairment of cognitive functions involving mainly executive functions and psychomotor retardation (plan and schedule actions, cognitive flexibility, attention, abstraction) there may not be focal signs (being rarely involved cortical areas), while frequent and persistent are non-cognitive consequences such as depression, apathy, gait disturbances, extrapyramidal signs and urinary disorders, that reduce quality of life<sup>(3,4,5)</sup>.

These events are probably caused, as we have seen, by interruption that ischemic lesions determine on prefrontal-subcortical circuits<sup>(6)</sup>. Among the

most common cognitive and behavioral impairment dominate decrease in activity and interest, apathy and inertia.

It was clear, therefore, that MID represents only a part, however large, of vascular dementia, not understanding different clinical and neuroradiological specific alterations. For these reasons, it was decided to replace term MID with diction “Vascular Dementia” (VaD), broader and flexible concept that embraced all clinical case secondary to ischemic or hemorrhagic encephalopathy. Vascular dementia would thus include MID and SIVD, as well as other less common forms such as single strategic infarcts dementia, hypoperfusion dementia, hemorrhagic dementia and genetic basis dementia (CADASIL).

Over time, however, knowledge improvement has made obsolete and imprecise criteria of VaD. Main limitations of VaD concept are to disregard numerous cases of dementia with both neurodegenerative and vascular etiology (the so-called “mixed forms”), the fact that all diagnostic criteria formulated required a mnesic deficit (being made on basis of diagnostic criteria for Alzheimer’s dementia), and finally they do not allow to identify frameworks of mild cognitive impairment<sup>(7)</sup>. Therefore Hachinski, in 1994 redefined boundaries of this nosological entity, both because term “vascular” was “too general” and because, for clinical purposes, diagnosis of vascular dementia in frank state was of little beneficial. He therefore proposed an alternative approach:

“Identify patients across the whole spectrum of vascular cognitive impairment, from high risk with no deficit (*brain-at-risk stage*) to full-blown dementia. Describe the cognitive impairment in terms of standardized neuropsychological measures, and relate the dementia to the specific vascular cause, so that the appropriate preventive measures can be implemented”<sup>(8)</sup>.

It is clear, therefore, the importance of expanding as much as possible spectrum of VaD, to include forms of cognitive impairment that exceed physiological senile deterioration without, however, meet diagnostic criteria for dementia, all the various subtypes of vascular dementia and finally forms of mixed origin. It is therefore coined the “umbrella” term Vascular Cognitive Impairment (VCI), that enclose a complex spectrum of disease entities, all associated by a common pathophysiological moment (brain damage of vascular nature, be it ischemic, hypoxic, or hemorrhagic) and characterized by dysexecutive syndrome and attentional

lability; memory disorder may be present but not prominent, and however different from amnesia of Alzheimer's dementia. With this approach we have therefore a greater chance of intervention, both in prevention and early therapy<sup>(4)</sup>, as this diagnostic concept allows us to identify high-risk patients already in preclinical phase, the so-called state of "brain at risk", renamed Vascular Cognitive Impairment-No Dementia: it comes to still autonomous subject but with a selective deficiency in a cognitive function, that it is important to detect early for purposes of performing a proper prevention correcting any vascular risk factors.

Even today, however, the concept of Vascular Cognitive Impairment is the object of numerous debates, proposals and revisions. Some authors<sup>(9,10)</sup> propose to reserve the term only to forms of VCI-ND without focal deficits (aphasia, apraxia, etc.), and to include more complex forms of vascular cognitive impairment in recent concept of Vascular Cognitive Disorder. In light of these further developments, the consensus of National Institute Neurological Disorders and Stroke and Canadian Stroke Network about VCI, concluded that latest guidelines represent the beginning of a new diagnostic process rather than end<sup>(11)</sup>.

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