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Artículo de Opinión

VASCULAR DEMENTIA: ITIS TIME FOR A NEW APPROACH

Demencia vascular, es tiempo de un nuevo enfoque

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Current concepts of vascular dementia are obsolete. Conventional definitions identify the patients too late, miss subjects with cognitive impairment short of dementia, and emphasize consequences rather than causes, the true bases for treatment and prevention.

Obsolescence of current concepts of vascular dementia:

For each patient considered demented, there is another individual with cognitive impairment short of dementia (CIND). The cornerstone of all criteria of dementia is memory impairment. This criterion works very well for Alzheimer's disease, where memory is an early and constant feature, but it seldom helps in identifying individuáis with cognitive impairment on a vascular bases. About 80% of all strokes occur in the carotid artery distribution, only 20% affecting the vertebrabasilar system, which supplies the hippocampi. While strokes in the medial temporal lobes :ause memory impairment, it usually takes bilateral lesions to cause serious and permanent memory problems. Strokes affecting cognition occur most commonly in the frontobasal systems that subserve judgement, planning and emotion, features seldom tested in cognitive screens. The most fundamental problem with current criteria of dementia is that they do not work. In the Canadian study of Health and Aging, which is both a population and institution based study, 1879 subjects were identified as demented by consensus. Then six commonly used criteria of dementia were applied to the same subjects. Little overlap emerged, and a surprising 10 fold difference separated the least and the most sensitive criteria. According to ICD-10 criteria, 3.1% of the population o ver the age of 65 years are demented, by DSM-III criteria 29.1% are!

Ascertainment and classification biases:

Most cliniopathological studies are carried out on patients who have been identified through memory or Alzheimerclinics. Since they come to attention because of memory problems, most of them will turn out to have Alzheimer's disease. By contrast, patients with cerebrovascular disease usually come to medical attention because they have had transient ischemic attacks or strokes. If these patients have cognitive impairment, it is often frontal lobe **dysfunction,** which **is**

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difficult to diagnose and test.

Even if identified, these patients seldom find their way into clinicopathological studies of dementia because the current definitions of dementia require memory impairment as the first requisite, and memory is seldom severely impaired in patients with cerebro vascular disease. Thusthere is a systematic overrepresentation of Alzheimer cases in clinicopathological series.

This is compounded by another phenomenon. Most clinicopathological studies claim an accuracy of about 90% in the diagnosis of Alzheimer's disease. However, only patients who die come to autopsy, typically 7-8 years after diagnosis in Alzheimer's disease.

If one includes in the denominator the 23% of patients who initially are diagnosed as Alzheimer's disease who do not deteriórate cognitively and survive, the accuracy drops to about two-thirds.

From multi-infarct dementia to vascular cognitive impairment:

We suggested the term "multi-infarct dementia" to emphasize that when dementia occurs on a vascular basis, it is usually due to múltiple infarcts and not to the slow strangulation of the brain's blood supply resulting in chronic cerebral ischemia and neuronal starvation and death.

While multi-infarct dementia occurs, it is rare in puré form. What is much more common is cognitive impairment on a vascular basis short of dementia. Thus, Bowler and I have suggested the term "vascular cognitive impairment" to describe the spectrum of cognitive impairment on a vascular bases, from mild impairment to frank dementia.

The commonest context of multi-infarct dementia occurring in patients with Alzheimer changes i.e. "mixed dementia". The "ischemic score" distinguishes very well puré Alzheimer's disease from multi-infarct and mixed dementia but not between the latter two. This does not matter pragmatically, the important point being the identification of the treatable vascular component.

Leukoaraiosis

We coined the term "leukoaraiosis" to describe white matter rarefaction as seen on brain imaging and to emphasize that although ischemia is often invoked as the cause, white matter changes may be due to múltiple causes. While white matter changes can occur in the elderly without any clinical correlates, consistent associations have been described between proventricular white matter changes and cognition. Subcortical white matter changes may be accompanied by depression. Leukoaraiosis is associated with an increased risk of stroke, vascular death, cognitive deterioration and with a 3-4 fold increased risk of intracerebral bleeding in patients placed on anticoagulants.'

Co-existence and possible interaction for stroke and Alzheimer's disease:

Snowden et al have shown that among elderly nuns having the pathological diagnosis of Alzheimer's disease only 57% were demented. Among those having the pathological diagnosis of Alzheimer's disease plus cortical infarcts 75% were demented and among those having pathological Alzheimer's disease and small infarcts in the subcortical áreas, 93% were demented. Clearly the effects of Alzheimer's disease and strokes add up or perhaps interact, since lesions in the brain do not add up, they multiply.

In a clinical pathological study addressing the question of education and dementia, no difference was found in the progression of dementia once diagnosed, among individuáis with a primary education, secondary education and college or university education. Símilarly, the degree of brain atrophy, presenceofsenile plaques and neurofibrillary tangles was

comparable among the educational groups. However, the Ieast educated patients had significantly higher prevalence of lacunar infarcts, white matter lesions, macroscopic infarcts and other vascular lesions than the other two groups with higher education.

The need for a new approach:

We need to take a pragmatic conceptual retreat before we are ready to advance at an accelerated pace. We need to identify potential patients long before they are demented, including symptomless individuáis at high risk of developing cognitive impairment. When-subjects have cognitive impairment, this should be characterized clinically and by a commonly agreed mínimum of standardized tests. We may have to make an initial diagnosis of a "cognitive syndrome" and become more precise as the data allow.

Conclusión

We should abandon current diagnostic categories and describe cognitive impairment clinically and according to commonly agreed instruments that document the demographic data in a standardized manner and undertake a systematic effort of identifying the underlying etiology in each case. Imaging and DNA should be obtained whenever possible.

An empiric approach and the use of mutually intelligible descriptions will contribute to a knowledge base that will increase not only our understanding, but wiíl give us the basis for therapy. Already, enough scientific rationale exists for undertaking systematic clinical triáis in the prevention of cognitive impairment through the control of vascular risk factors and the use of statins, anti-inflammatory agents and ACE inhibitors. We need to overturn our current approach of gathering data into arbitrary, dogmatic, obsolete categories and begin developing a new pragmatic, systematic, data based approach.

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