Vascular dementia

Vascular dementia (VaD) is the second most common form of dementia after Alzheimer's disease (AD), and one of the major causes of mental and physical disability in developed countries. As such, the identification and implementation of strategies which prevent the development of the condition or enable improvements in patients with VaD are healthcare objectives of the first order. VaD is now regarded as a combined group of clinical-pathological entities rather than one disease, that is, multiple pathogenic mechanisms and lesion types underlie a cognitive impairment of vascular origin.

The clinical diagnosis of VaD is complex and difficult because of the heterogeneous nature of its clinical presentation and progression and the low sensitivity of existing clinical criteria. Moreover, there is growing evidence of the epidemiological significance of mixed forms of dementia, and that ischemic processes may precipitate and exacerbate cognitive impairment in AD.

Numerous compounds have been proposed as potentially useful in the treatment of patients with VaD, comprising vasodilatative, antithrombotic, hemorrheological, nootropic, antiserotoninergic and, most recently, antiglutamatergic and cholinergic approaches.

In spite of some initially favorable reports based on the use of memantine, donepezil and galantamine, there is as yet no conclusive evidence of a definitive treatment for VaD.

Unsatisfactory results from VaD drug trials may be attributed in part to the diversity of the patients included (underlying pathogenic mechanisms, number, type, and location of vascular lesions), and to methodological limitations in the design of the trials (outcome measures, end-points, size, follow-up period).

The treatment of modifiable vascular risk factors - hypertension, diabetes mellitus, hypercholesterolemia and heart disease - is an

important strategy for the reduction of the risk of dementia, and is likely to slow the progress of cognitive decline.