Chapter 7
Stroke:
A Potential Risk Factor of Neurodegenerative Disorders

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ABSTRACT

The brain relies on a specialized endothelial system, the blood brain barrier (BBB), which is capable of regulating the transfer of substances from the blood to the neurons. Stroke is the most frequent cause of disability in adulthood. Lesions of vascular origin also include asymptomatic small infarcts, microbleeds, dilated perivascular spaces, and atrophy. Vascular cognitive impairment (VCI) is the second most prevalent cause of dementia. Several mechanisms are implied, including strategic infarct dementia, post-stroke dementia, cerebral amyloid angiopathy, and subcortical vascular dementia. As there is no disease modifying therapies currently available, treatment of comorbidities and adequate control of the vascular risk factors remain the standard strategies to reduce the vascular contributions to neurodegeneration. This chapter represents the basic concepts of pathophysiology of cerebrovascular diseases, and describes the subtypes of VCI, as well as treatment and primary prevention strategies.

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INTRODUCTION

There is no single mechanism that satisfactorily explains the complex relation between stroke and neurodegenerative disorders, predominantly those related to cognition impairment. Rather, this relation may be explained by multiple pathophysiological mechanisms, such as microstructural defects, leading to blood brain barrier and neurovascular unit dysfunction; by vascular mechanisms, when traditional vascular risk factors lead to failure on the capacity of cerebral blood flow regulation; and by genetic predisposition, exemplified by CADASIL, apolipoprotein ε polymorphisms and variations of homocysteine´s metabolism. Furthermore, these are not necessarily exclusive, and a combination of these factors may occur in individual patients (Iadecola, 2013; Schneider *et al*, 2003).

In patients with cerebrovascular disease, cognitive decline, affecting mainly executive domains and language functions, may occur. A variety of cerebrovascular conditions will result in distinct profiles of cognitive impairment, by either lowering the threshold for neurodegenerative diseases or unmasking an already present, often mild or subclinical, dementia.

Vascular cognitive impairment (VCI) is a condition in which vascular lesions can lead or contribute to impaired cognitive function, and ranges from mild cognitive disorder to dementia (Bowler and Hachinsky, 1996; Gorelick *et al*., 2011). It is the second most prevalent cause of cognitive impairment, and remains vastly underdiagnosed (Godefroy *et al*., 2013).

There are several clinical subtypes of VCI, such as poststroke dementia, with a short-term incidence after stroke as high as 40% (Pendlebury and Rothwell, 2009); strategic infarct dementia, caused by interruptions of strategic networks, leading to impaired executive functions; subcortical vascular dementia, by far the most common subtype of VCI; microinfarcts dementia, caused by small lesions detected only by high resolution brain imaging or biopsy; microbleeds related VCI, which could be caused either by hypertensive vasculopathy or cerebral amyloid angiopathy; hypoperfusion dementia, as a consequence of hemodynamic failure in border zone (watershed) regions; and mixed disease, when vascular pathology and Alzheimer Disease (AD) overlap.

In this context, VCI appears as a continuum of different clinical entities, which makes it the perfect prototype to understand the fascinating connection between cerebrovascular disease and neuronal degeneration (Iadecola, 2013; Schneider *et al*, 2003).

This chapter review the concepts of blood brain barrier, neurovascular unit and brain autoregulation; define stroke and its subtypes (ischemic, hemorrhagic and cerebral venous thrombosis), with their main risk factors, clinical features and morbidity; explain the impact of cerebrovascular diseases on cognition and neurodegeneration; and describe the subtypes of VCI, as well as treatment and primary prevention strategies.

BACKGROUND

The attempt to divide normal ageing from pathological senility began in the late 19th century, which was a critical period for developing the concept of dementia as we know it today. At that time, the definition of dementia was similar to the one used nowadays, although much more inclusive: a general compromise of intelligence, with prominent memory loss and alterations of personality. Studies by French and German psychiatrists were crucial to separate psychotic syndromes, such as schizophrenia, from other forms of cognitive impairment. This interest in understanding pathological aging has encouraged a great number