FOREWORD

Post-ischemic brain damage is one of the leading causes of death worldwide, resulting in increased disability that significantly impacts patients' quality of life, healthcare costs, and resource use. The incidence of cerebral ischemia increases with age, and it is estimated that about 20% of cases occur in young people between the ages of 18 and 50. The incidence of cerebral ischemia has increased in the younger population under the age of 55, especially in Europe and the United States. However, in the elderly population, the risk of cerebral ischemia is 1 in 3. Currently, approximately 17 million patients each year suffer from ischemic brain injury, of which 6 million will die. In addition, long-term complications, such as the development of dementia, can affect up to half of all survivors of cerebral ischemia and they can live with the debilitating consequences of ischemia for more than twenty years.

Recently, significant advances in knowledge of cerebral ischemia have been observed through the use of proteomic and genomic tools in laboratory studies of post-ischemic brain neurodegeneration with the Alzheimer's disease phenotype and genotype. In clinical and experimental studies following cerebral ischemia, a characteristic neuropathology of Alzheimer's disease was found, including diffuse and senile amyloid plaques and pathology of the tau protein. In the post-ischemic brain, increased production and accumulation of amyloid in affected areas, such as the hippocampus, and accumulation of amyloid in the vessel walls in the form of cerebral amyloid angiopathy have been found. Moreover, the observed hyperphosphorylation of the tau protein with the development of neurofibrillary tangles suggests that it plays a key role in the pathogenesis of post-ischemic brain neurodegeneration. In an experimental model of cerebral ischemia, the genes associated with Alzheimer's disease were found to be dysregulated, i.e., the amyloid protein precursor, β-secretase, presenilins and the tau protein. Consequently, cerebral ischemia is now recognized as the leading cause of Alzheimer's disease dementia or Alzheimer's disease. The data indicate that in experimental post-ischemic brain injury, amyloid and the tau protein further contribute to neuronal death, along with the development of neuroinflammation. Therefore, it is extremely important to focus on all these new aspects of post-ischemic brain changes to improve patient outcomes. In this situation, treatment after cerebral ischemia requires a multidisciplinary team with extensive clinical experience to provide adequate individual treatment to eligible patients, with the implementation of new rehabilitation strategies for recovery. To help with rehabilitation after ischemia, research is underway to use stem cells to improve neurological outcomes. This book is of great value to a wide audience looking for new information on cerebral ischemia, such as clinicians, and indicates potential new areas of research for scientists and gives the reader an insight into what is on the horizon.

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