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Prevention and Management of the Neurodegenerative Diseases

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Introduction

Alzheimer's disease is the most common neurodegenerative disease. It also represents the most frequent cause of dementia, accounting for roughly half of all cases. The prevalence of AD is roughly 30% among people 85 years and older. Incidence rates climb steeply from 0.5% per year for ages 65 to 75 to 6–8% per year for ages 85 and up. AD onset is rare before the age of 50, except in cases of familial Alzheimer's disease, which comprise roughly 5–10% of cases [1]. The primary clinical manifestation of Alzheimer's disease is dementia, which is an accelerated loss of cognitive function beyond that due to normal aging. Alterations in mood and behaviour often accompany the onset of dementia, followed by memory loss, disorientation and aphasia. The hippocampus and cerebral cortex are preferentially and severely affected in Alzheimer's disease. Pathologically, senile or neuritic plaques and neurofibrillary tangles are the two characteristic lesions in affected tissues [2]. Neuritic plaques in blood vessels and neurons of the hippocampus are primarily composed of amyloid β peptide aggregates. The second pathological hallmarks, neurofibrillary tangles, are filamentous bundles comprised of abnormal tau proteins that accumulate in the cytoplasm of affected neurons [3]. Tau protein is normally involved in nutrient transport along neuronal axons. Various lines of evidence indicate that Alzheimer's disease develops primarily as a result of an amyloid cascade. Aggregation of hyper-phosphorylated tau proteins leading to tangles may also contribute to this cascade mechanism [4]. Other potentially relevant disease mechanisms include, micro-vascular damage, leading to diminished blood flow and nutrient eficiency to brain cells; oxidative stress; inflammation; and mitochondrial dysfunction. Family studies have established that genetic factors play a substantial role in Alzheimer's disease, especially in younger onset cases. Familial Alzheimer's disease has an autosomal dominant inheritance pattern [5]. Three mutations in genes encoding proteins involved in amyloid plaque formation, the amyloid precursor protein, presenilin-1, and presenilin-2 genes, have been identified as causal genes for early-onset Alzheimer's disease [6]. Non-familial Alzheimer's disease, typically defined as having an onset at age 65 years or older, accounts for most of cases. Non-familial Alzheimer's disease has been associated most consistently with the ε4 allele of the apolipo protein gene, which is a very low-density lipoprotein carrier that is required for Aβ deposition [7]. Carriers of the ApoE-ε4 allele have reduced Alzheimer's disease ages at onset, with 3-fold and 15-fold risk excesses observed in heterozygotes and homozygotes, respectively. Numerous other candidate genes have been investigated as Alzheimer's disease susceptibility factors, such as sortilin-related receptor-1 gene, but no strong or consistent findings have emerged. Increasing age is a clear risk factor for non-familial Alzheimer's disease, and rates are generally higher in women than in men [8]. Other factors that have been investigated in relation to Alzheimer's disease risk include: cardiovascular diseases, head trauma, smoking, dietary antioxidants and fats, alcohol, occupational exposures to solvents, electromagnetic fields, educational status, and occupational exposures to pesticides. Epidemiologic evidence has been mixed thus far, as exemplified by contradictory findings for cigarette smoking [9]. It is possible, yet remains to be established conclusively, whether genetic factors account for the majority of the population attributable risk for Alzheimer's disease. Parkinson's disease, the second most common neurodegenerative disease, is a movement disorder whose cardinal clinical features are rest tremor, rigidity, bradykinesia and postural instability. Parkinson's disease is relatively rare before age 50, after which incidence and prevalence rise sharply through the eighth decade of life. Epidemiologic surveys, mainly in western countries, indicate a small excess risk in men. Annual incidence rates of 10–15 per 100 000 have been noted in most surveys worldwide [10]. Prevalence may reach 2% in persons aged 65 years and older. The underlying cause of Parkinson's disease is a loss of dopamine-producing neurons of the mid-brain substantia nigra. Parkinson's disease pathogenesis involves complex interactions among several mechanisms, including abnormal protein aggregation and deficient clearance of aggregates, altered dopamine metabolism, impaired mitochondrial function, oxidative stress, inflammation, necrosis and accelerated apoptosis.

Acknowledgement

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Conflict of Interest

None

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