ABSTRACT

Alzheimer’s Disease (AD) is a chronic progressive and irreversible neurodegenerative brain disorder. The aging population has been increasing significantly in recent decades. Therefore, AD will continue to increase because the disease affects mainly the elderly. Its diagnostic accuracy is relatively low, and there is not a biomarker able to detect AD without invasive tests. The electroencephalogram (EEG) test is a widely available technology in clinical settings. It may help diagnosis of brain disorders, once it can be used in patients who have cognitive impairment involving a general decrease in overall brain function or in patients with a located deficit. This study is a new approach to detect EEG temporal events in order to improve the AD diagnosis. For that, K-means and Self-Organized Maps were used, and the results suggested that there are sequences of EEG energy variation that appear more frequently in AD patients than in healthy subjects.

INTRODUCTION

In the last decades there have been deep changes in the age structure of the world population, characterized by a progressive decrease in the young population and inversely, by an increasing proportion of elderly people (Ballard, et al., 2011). The decrease of mortality and the simultaneous decrease of fertility levels have contributed to the phenomenon called overall aging of the population. The aging population has become a fact of scientific interest around the world because the elderly are most vulnerable to the onset of certain degenerative diseases (Jonker, Launer, Hooijer, & Lindeboom, 1996). Alzheimer’s disease (AD) is one of the most debilitating diseases of developed societies. AD is the most common cause of dementia and in 2001 reached more than 21.1 million of affected people around the world (Ballard, et al., 2011). AD represents 60% of elderly diseases, and it can be understood as a cortical
and degenerative brain disorder, what means that gradually, over time, most parts of the brain will suffer damage and symptoms will severely increase (Blennow, Leon, & Zetterberg, 2006). AD has an unknown cause. The first symptoms of AD are memory loss, particularly difficulty remembering newly learned information, and concentration problems (Gwyther, 2001). As the disease progresses patients manifest general cognitive problems, confusion, personality / behavioural changes and also disorientation (Bird, 2001). Finally, there is a global brain atrophy and patients acquire a complete inability (Cummings, 2004). So, permanent aids of family members or caregivers are increasingly requested (Jeong, 2004).

The progression of AD is gradual and is usually categorized in four stages. Each of these stages has typical symptoms of the disease progression. The pre-dementia stage is known as Mild Cognitive Impairment (MCI), and it is characterized by subtle symptoms where cognitive deficits are only limited to memory and patients life activities are preserved (Nestor, Scheltens, & Hodges, 2004). The MCI is considered a transitional stage between the normal aging and AD. The MCI confers an increased risk of developing AD but only between 6% and 25% of people affected with MCI progress to AD (Shimokawa, et al., 2001). The next two stages of AD are Mild and Moderate AD which are typically characterized by a significant increase of cognitive deficiencies and a marked loss of independence (Gwyther, 2001). The last stage of the disease is Severe AD, in which a complete deterioration of personality occurs, and patients depend entirely on help from caregivers (Mesulam, 2000).

Senile plaques and neurofibrillary tangles in the medial temporal lobe and cortical areas are two pathological hallmarks of brains ravaged by AD (Blennow, 2005; Mattson, 2004). These two abnormal structures are responsible for damaging and killing nerve cells (Cummings, 2004). Parts of the brain start to shrink because the brain’s nerve cells die. In the last stage of AD, damage is generalized, and the brain tissue has decreased significantly in volume (Blennow, 2005; Blennow & Zetterberg, 2010).

Experts do not know yet why the brain cells deteriorate. Several factors are often associated with a higher risk of developing AD. Ageing is considered the main risk factor to acquire AD; after the age of 65, the risk of developing the disease doubles every five years. Another factor is family history; people who have a close relative who developed Alzheimer’s disease have a slightly higher risk of eventually developing the disease themselves (Blennow, 2005). Gender is considered another factor; a higher percentage of women develop AD than men. Heart disease, like high cholesterol, hypertension or poorly controlled diabetes are considered risk factors to develop AD. Many other factors, including stress, obesity, smoking, lower educational qualifications, Down’s syndrome and head injuries are associated to a possible risk of developing AD (Blennow, Leon, & Zetterberg, 2006; Lahiri, Farlow, Greig, & Sambamurti, 2002).

Despite the progress in better understanding the AD stages, there remains no prospect of cure at least in the near future. Therapies currently available only soften and slow the progression of symptoms that is why new treatments must be developed to alter the disease process itself and not just to reduce some of the common symptoms (Ballard, et al., 2011). AD has been researched for years but continues to challenge medical research, and researchers have not yet discovered a good treatment for this complex and stressful disease and neither have discovered a reliable method to make the correct diagnosis of AD (Ballard, et al., 2011).

AD leads to death approximately seven years after being diagnosed (Mölsä, 1986). An early and accurate diagnosis is necessary to help intervention in order to reduce the brain damage. It is often difficult to diagnose AD and generally, diagnosis is made by excluding other possible causes of dementia symptoms (Porth & Matfin, 2009). Besides, a definitive diagnosis of AD can only be made by necropsy (Blennow, 2005). The development of methods that can
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