

Probable Effect of Alendronate in Alzheimer Disease, Tracking with MTR Imaging

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ABSTRACT

Alzheimer's disease (AD) is a neurodegenerative disorder. osteoporosis is one of the most common complications observed in AD patients. Magnetization transfer is an image contrast improving technique in magnetic resonance (MR) imaging (i.e., magnetization transfer ratio [MTR]). The perturbation of brain cholesterol homeostasis is involved in several neurodegenerative diseases, including AD. As several animal studies has been shown that alendronate has an effect on lowering cholesterol in CNS, consequently improving signs and symptoms of AD, we suggest prescribing a trial of alendronate in AD patients using MTR imaging to determine the effect of treatment.

Introduction

Alzheimer's disease (AD) is a chronic progressive neurodegenerative disorder. Based on the disease progress, symptoms vary from memory loss to severe dementia [1]. AD classically presents with gray matter atrophy, as well as significant white matter abnormalities [2]. Osteoporosis such as Alzheimer disease, is a multifactorial progressive degenerative disorder, which can cause weakening of bones in the body and increase risk of broken bones. osteoporosis is a common complication observed in AD patients [3]. Magnetization transfer is an image contrast improving technique in magnetic resonance (MR) imaging (i.e., magnetization transfer ratio [MTR]). MTRs can be used to detect changes in the structural status of brain parenchyma that may or may not be visible with standard MR techniques. These findings may be due to myelin loss, chronic edema, or previous undetected tumor [4].

Amyloid precursor protein (APP) processing and subsequent A β production are dependent on membrane cholesterol content and on levels of isoprenoid intermediates in the cholesterol biosynthesis pathway, changes in cholesterol might have different consequences on A β formation [5]. The perturbation of brain cholesterol homeostasis is involved in several neurodegenerative diseases, including AD, and could thus represent a major therapeutic target [6]. The relationship between vascular diseases and AD highlights the importance of high cholesterol levels in the development of AD [7]. Cholesterol-modifying agents offer a safe and readily available alternative as it involves in the pathophysiology of AD.

Current therapeutic strategies for AD focus on cognitive deficit alleviation solely via direct AChE inhibition [8]. Alendronate suppresses AChE activity in frontal cortex (the site of the highest A β accumulation). A study in rats, applying Alendronate, revealed alendronate significantly decreased the activity of AChE in the frontal cortex, no change was seen in other parts of the brain [9]. Another study resulted that alendronate treatment in rats decreases cholesterol biosynthesis in CNS. Build-up of A β peptide is associated with a reduction of cholinergic transmission, which is characteristic for Alzheimer disease. Since other cholesterol-lowering drugs (statins) play a putative preventive role in AD [10]. Statins, the common indication in AD for control the progress or postpone the onset, has a pathology on decreasing cholesterol in blood. As several animal studies has been shown that alendronate has an effect on lowering cholesterol in CNS, consequently improving signs and symptoms of AD, we suggest prescribing a trial of alendronate in AD patients, withal, we offer using MTR imaging to determine the effect of treatment.

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